CLINICO-PATHOLOGICAL STUDY OF DIABETIC FOOT IN BUNDELKHAND REGION

[A CONTINUED STUDY]

THESIS

FOR MASTER OF SURGERY

(GENERAL SURGERY)

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ANURAG SRIVASTAVA

Dedicated to

My teachers, My Parents,

My Friends & GOD

DEPARTMENT OF SURGERY
M.L.B. MEDICAL COLLEGE
JHANSI

CERTIFICATE

This is to that the work entitled "**CLINICOPATHOLOGICAL STUDY OF DIABETIC FOOT IN BUNDELKHAND REGION**" which is being presented as a Thesis for M.S. (Surgery) Examination, 2007 of Bundelkhand University; Jhansi has been carried out by **Dr. Anurag Srivastava** in the Department of Surgery, M.L.B Medical College. Jhansi. This study has been conducted under my direct supervision and guidance.

The observations recorded were periodically checked and verified by me.

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DEPARTMENT OF MEDICINE
M.L.B. MEDICAL COLLEGE
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This is to that the work entitled "**CLINICOPATHOLOGICAL STUDY OF DIABETIC FOOT IN BUNDELKHAND REGION**" which is being presented as a thesis for M.S. (Surgery) by **Dr. Anurag Srivastava** was conducted under my direct supervision and guidance. The observations recorded were periodically checked and verified by me. This work fulfills the basic ordinances governing the submission of thesis laid down by Bundelkhand University.

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Dated 13/12/2006

DR.ANURAG SRIVASTAVA

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INTRODUCTION

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Diabetes was first described in chronicles II of the BIBLE when King Asa developed gangrene of the feet. In 1887, Pryle described painful symmetrical polyneuropathies both clinically & pathologically in a diabetic patient. Charcot described pronounced leg weakness, with ataxia, which he termed ***"Diabetic Paraplegia".***

Diabetes Mellitus is a disease of antiquity. There is mention of symptoms & treatment of this disease by ***Eben Papyrus (1500 B.C.)***. An ancient Hindu document by ***Susurta (400 B.C.)*** described the diabetic syndrome as characterized by ***"Honeyed Urine"***.

Diabetes mellitus is defined as an endocrine disease resulting from either a deficiency or release of insulin into blood stream; or a resistance to the insulin, produced in the body. At present about 150 million people have diabetes mellitus & the prevalence is rising, particularly in developing world. Indeed, by year 2010, it is estimated that there will be approximately 250 million people worldwide with diabetes mellitus ***{Fast Facts-Diabetes Mellitus-Oxford-Campbell I.W., Lebovitz Harold, 2000}***.

The greatest fears of the diabetic patients are loss of eye sight & amputations.

The diabetic complications the most common cause of at risk foot & is the reason for the majority of non traumatic lower limb amputations ***(Merriman & Tollafield, 1995),*** due to frequent complications of peripheral neuropathy, infections & Peripheral vascular disease [P.V.D.].

Diabetic foot is a clinical syndrome involving pain, deformation, inflammation, infection, ulceration & tissue loss of the foot in a diabetic patient.

The incidence of diabetic foot as a diabetic complication is more than the combined incidence of retinopathy, nephropathy, MI and Stroke.

Among people with diabetes 15% will have a foot ulcer in their life time.

Indeed about 14-24% of people with a foot ulcer will require an amputation. In United States, the annual incidence of lower extremity, leg and foot ulcer is 200,000 and 56,000 major amputations are done for them. This is greater than all the either complications of diabetes including coronary artery disease (10100), stroke (27000), blindness (6900) & renal failure (5900).

In Australia nearly half of all leg amputations are attributed to complications resulting from diabetic foot problems, mainly peripheral neuropathy ***{Short & Charmichael, 1998}***.

More than 10% of all admissions for diabetes are primarily for foot management at the ***Indian Institute of Diabetes in Bombay***. More than 70% required surgical intervention & in more than 45% of these interventions were toe or limb amputation.

In UK, more than 50% of bed occupancy of diabetes is due to toot problem.

So, it is obvious from those figures that throughout the world diabetic foot problems are a major cause of hospitalization morbidity.

In India, prevalence of diabetic mellitus in adults was found to be 2.4% of rural & 4-11.6% in urban dwellers. High frequencies of impaired glucose tolerance ranging from 3.6-9.1 indicate the potential further rise in prevalence of diabetic mellitus in coming years.

Persons with diabetic are at significant risk for lower extremity amputation. A person with diabetes is 15 times more likely to have a lower extremity removed than a non-diabetic individual; about half of these amputees will develop a

limb threatening condition of the contra-lateral limb within 18 months and will require amputation in 5 to 8 years. ***Pecoraro and colleagues*** surveyed 80 consecutive diabetic foot amputations and found that and initial episode of minor trauma that resulted in cutaneous ulceration with subsequent failure to heal the wound preceded 72% amputations. It is well known fact that diabetic foot problem needs special attention if there is underlying neuropathy, ischemia or uncontrolled hyperglycemia.

Foot ulcer & infections are a major source of morbidity in individual with diabetes mellitus. The reasons for the increase incidence of these disorders, in the diabetes mellitus are complex & involve interaction of several pathogenic factors: i.e. neuropathy, abnormal foot biomechanics, P.V.D. and poor wound healing impede resolution of minor breaks in the skin, allowing them to enlarge & become infected.

Risk factors for foot ulcers or amputations in DM include – male sex, diabetes > 10 year duration, peripheral neuropathy, abnormal structure of foot (bony abnormalities, callus, thickened nails), PVD, smoking & history for previous ulcer or amputation. Glycemic control is also a risk factor, each 2% increase in the HbA^{IC} increases risk of lower extremity ulcer by 1.6 times and risk of lower extremity amputation by 1.5 times.

The focus of this study will be on the clinical & pathological events related to the development of diabetic foot syndrome in diabetic patients with special reference to management of diabetic foot problems.

Bundelkhand area, having a rich heritage and culture, is socio economically backward part of this country and is population is about 88, 32,987[Jan 2001].

REVIEW OF LITERATURE

REVIEW OF LITERATURE

DM is defined as an endocrine disease resulting from either a deficiency in the production or release of insulin in to the blood stream; or a resistance to the insulin produced in the body. Diabetes is a chronic disease characterized by lack of insulin or significantly reduced effectiveness of insulin. The normal limit of blood glucose level within the body is 3.5 - 8.0 mmol/L ***(Levin O'Neal & Bowker, 2003).***

CLINICAL CLASSIFICATION OF DIABETES MELLITUS:

(WHO adopted)

1. Diabetes Mellitus
   I) Insulin Dependent Diabetes Mellitus [IDDM, type 1).
   II) Non-insulin dependent diabetes mellitus (NIDDM,type2).
   iii) Malnutrition related diabetes mellitus (MRDM).
   iv) Other type (Secondary to pancreatic hormonal, drug induced, genetic and others).
2. Impaired glucose tolerance (IGT).
3. Gestational diabetes mellitus (GDM).

Experts from National Diabetes Data group & WHO gave the criteria for diagnosing Diabetes Mellitus as -

-Symptoms of diabetes+ random blood glucose conc. ≥11.1mmol/L {200mg/dl}

-Fasting plasma glucose > 7mmol/L (126mg/dl).

-Two hour plasma glucose>11.1 mmol/L (200mg/dl) during an oral glucose tolerance test.

Pathophysiology of Diabetic Foot

Foot problems in patients with diabetes mellitus are a major public health concern in the United States. In 1990 the national Centers for Disease Control

estimated there were 14 million people in the United States affected by diabetes of which an estimated 25 percent will develop foot problems. Presently, foot problems account for 20 percent of the annual diabetic-related hospitalizations. More than 50 percent of the 120,000 non-traumatic, lower-extremity amputations each year result from complications of diabetes. Neuropathy, mechanical stresses and angiopathy (ischemia) are the major causes of foot ulcers in diabetic patients; however, a number of other factors have been cited.

Two major factors contribute to the problems encountered by the person with diabetes: Peripheral polyneuropathy and macro vascular disease. It is no longer acceptable to blame diabetic foot problems only on microangiopathy. Infection in presence of polyneuropathy and or ischemia does all the damage.

1. NEUROPATHY

Altered nerve metabolism resulting from chronic hyperglycemia is the likely cause of polyneuropathy in diabetes.

A distal, mixed sensory-motor-autonomic neuropathy is most common, involving both the large- and small-diameter fibers. There is a predominance of sensory over motor involvement.

Loss of pain and temperature sensation predisposes the area of involvement to repeated injuries such as burns, abrasions or mechanical stresses. Distal motor neuropathy results in weakness of the foot's intrinsic muscles, leading to the development of claw toe and cavus foot deformities. Weakness of extrinsic peroneal nerve muscles contributes to equinovarus deformities. These deformities cause an abnormal weight bearing distribution.

Loss of pain sensation is widely accepted as the primary cause of ulceration of the diabetic foot.

Brand demonstrated this important concept in the development of plantar ulceration in leprosy and diabetes patients. ***Boulton et. al.*** found diabetic

patients with plantar ulceration had significantly decreased vibratory sensation and increased plantar pressure compared to diabetic patients without ulceration or normal controls.

In a recent study of the causal pathways in lower-extremity amputation of 80 diabetic patients, sensory loss was found in 82 percent of the cases while ischemia was found in 46 percent.

Loss of Protective Sensation:

Assessing sensory loss by various means has been well studied. ***Sosenko et. al.*** compared sensory testing with pressure, vibratory and thermal measures and found pressure thresholds using nylon filaments are the most sensitive and specific. Several studies support the use of the ***10-gram (Semmes-Weinstein 5.07) nylon filament*** as the threshold for protective sensation. Patients unable to feel a 10-gram nylon filament are considered unable to protect their feet from injury and are at risk of ulceration. Patients with loss of protective sensation should be properly fitted with footwear designed to protect the foot and reduce stresses.

The contribution of autonomic neuropathy in foot ulceration has not been well studied, but it may be a factor in both ulceration and faulty healing of such. Loss of sweat gland function may result, allowing ulcer development due to dry, cracked skin.

Sympathetic denervation results in dilation of the arteries and arterioles increasing blood flow to the foot. This condition is associated with arteriovenous shunting that rushes blood from the arterial to the venous side of circulation, thus bypassing the capillary nutrient circulation. Long-term sympathetic denervation may cause structural changes in the artery and lead to medial wall calcification. Reduced capillary flow may increase the tissue susceptibility to injury, slow tissue healing and reduce tissue resistance to infection.

Autonomic neuropathy also may result in loss of the veni-vasomotor reflex. This reflex controls rises in venous pressure, especially during standing, by increasing precapillary resistance to blood flow. Loss of this reflex causes increased venous pressure and pooling, which promotes tissue edema. Edema

can be a complicating factor in wound healing.

The dilation and shunting of vessels increases the blood supply to the bones of the foot. Bone scan studies with radio-pharmaceutical agents showed increased uptake proportional to increased blood flow and osteoblastic activity in neuropathic patients compared to non-diabetic controls. *Accelerated osteoblastic activity results in demineralization and predisposes the bones to damage (Charcot osteo-arthropathy) by minor trauma.* Loss of pain sensation from sensory neuropathy lets minor trauma occur.

In summary, *autonomic-neuropathy causes loss of sweating: rigid, dilated arteries; and arteriovenous shunting. These symptoms result in dry skin, relative distal ischemia, edema, demineralization of bone and increased blood flow to the foot while bypassing the capillary nutrient circulation.* In general, the foot faces greater risk of injury and infection, and healing may be impaired.

Characteristics of Neuropathic Ulcers:

Neuropathic ulcers are generally painless, round, surrounded by callus and located over prominent bony areas of the toes or plantar surface of the foot. There may be multiple lesions, but usually there is just one. The most common sites of ulceration are the first metatarsal head and the plantar aspect of the great toe. The foot is warm, dry and pink. The patient is initially unaware of the lesion and only notices it by the presence of blood or pus. Loss of sensation is an essential predisposing factor accompanied by mechanical, thermal or chemical injury.

Mechanisms of Injury:

Brand described three mechanisms of injury in the neuropathic foot: ischemia, direct trauma and repetitive stress. Ischemia occurs when blood flow to the tissues is blocked by low pressures (1 to 5 psi) over long periods of time, Ischemic injury is most commonly caused by wearing tight shoes. Direct trauma results from a single high pressure greater than 1,000 psi and only occurs if a

patient walks barefoot on a sharp object or a nail penetrates a shoe. *The most common cause of injury is repetitive stress.*

Moderate pressures (about 20 psi) repeated thousands of times a day can cause ulceration. Feet are subjected to similar repetitive stresses during walking. A person with normal sensation may develop inflammation from repetitive walking stresses, but pain will cause him to remove the source of irritation, change the way he walks or stop the activity.

The person with loss of protective sensation, however, continues to walk in the same manner, unaware of impending injury. Plantar ulcerations occur at the sites of highest pressure, and these loads are significantly higher in ulcerated-as compared to non-ulcerated-feet. ***Stokes et al.*** measured load under the feet of normal subjects and diabetic patients using a force plate.

No differences in force due to age or sex were found within the normal. Maximal loading was increased in diabetic patients with ulcers compared to those without ulcers and normal. The position of maximal loading corresponded to the site of ulceration with greater than normal loading corresponding to callus sites.

There was an association between body weight and loading. Diabetic patients with ulcer had decreased loading on the toes compared to normal. ***Ctereteko et al,*** studied forces on the feet of diabetic patients with ulceration, those with neuropathy but no ulceration, and normal subjects while walking on a load-sensitive platform. Their findings supported those of ***Stokes et al.*** *Toe loading was found to be decreased in diabetic patients compared to normal, and the site of maximum force was found under the site of ulceration.* Ulcerated patients were also heavier than those without ulceration.

Cavanagh et al., using a pressure platform, also found the site of ulceration in diabetic patients corresponded to the location of highest

pressures on the foot and confirmed the decrease of toe loading in diabetic patients. They concluded structural deformities resulted in areas of abnormally high pressure and recommended pressure assessment as part of routine foot screening in the early stages of the patient's disease.

When deformities are found in the presence of neuropathy or peripheral vascular disease, the foot is at a high risk of ulceration. Ulcers may be prevented by orthoses and modified footwear designed to reduce foot deformity-induced areas of high pressure.

A number of factors contribute to the development of areas of high loading on the foot, including body weight, deformity and hypo-mobility. ***Gibbs and Boxer*** described the relationship of biomechanical deformities of feet and hyperkeratosis .They noted the most common biomechanical abnormalities were rear foot varus, forefoot varus, rigid plantar flexed first ray and equinus. Rear foot and forefoot varus were causes of hyperkeratosis along the lateral and plantar aspects of the forefoot in the foot lacking compensatory pronation.

In the varus foot with compensatory pronation and normal dorsiflexion mobility of the first ray, hyperkeratosis forms on the middle three metatarsal heads. When the first ray is rigid, however, keratosis will develop over the first and fifth metatarsal heads. Hyper mobility of the first ray into dorsiflexion results in abnormal pressure on the medial aspect of the great toe and a "pinch callus" develops. Equinus results in increased pressure under all metatarsal heads because tightness of the Achilles tendon forces patients to walk on the balls of their feet. In patients with diabetes mellitus and loss of protective sensation, these deformities may cause ulcers and, eventually, deep sinus tracts.

Orthoses designed to balance the foot with biomechanical deformities, and thereby reduce mechanical stresses, have been recommended. Studies are

needed to show the effectiveness of biomechanically designed orthoses in reducing pressures. By studying the effect of barographic pressure on nondiabetic patients, Lang-Stevenson et al. found that high pressures over the area of healed ulcerations were reduced by surgical correction of deformities.

Charcot Deformities:

The term Charcot foot refers to bone and joint destruction that occurs in the neuropathic foot ***(Sanders & Erykberg, 1991)***. It can be divided into three phases.

1. Acute onset.
2. Bony destruction / deformity.
3. Stabilization.

1.*Acute Onset*

The foot presents with unilateral erythema, warmth and oedema. There may be a history of minor trauma. About 30% of patients complain of pain or discomfort. X-ray at this time may be normal.

However, a Tc-99m diphosphonate bone scan will detect early evidence of bony destruction.

Early diagnosis is essential. Cellulites, gout and deep vein thrombosis may masquerade as a Charcot foot. Initially the foot is immobilized in a non-weight bearing cast to prevent deformity. After one month a total contact cast is applied and the patient may mobilize for brief period. However the patient is given crutches and encouraged to keep his walking to a minimum ***(Selby et al, 1994).***

2. *Bony Destruction*

Clinical signs are swelling warmth and deformities which include the rocker bottom deformity and the medial convexity. X-ray reveals fragmentation fracture, new bone formation, subluxation and dislocation.

The aim of treatment is immobilization until there is no longer evidence on

x-ray of continuing bone destruction and the foot temperature is within 2℃ of the contraleteral foot. Deformity in a Charcot foot can predispose to ulceration which may become infected and lead to osteomyelitis.

This may be difficult to distinguish from neuropathic bone and joint changes; as on X-ray, bone scan or magnetic resonance imaging appearances may be similar. However if the ulcer can be probed to bone osteomyelitis is the more likely diagnosis.

3. Stabilization

The foot is no longer warm and red. There may still be opened but the difference in skin temperature between the feet is less than 2°C. X-ray shows fracture healing sclerosis, and bone remodeling. The patient can now progress from a total contact or Aircast to an orthotic Walker fitted with cradled moulded insoles. However, too rapid mobilization can be disastrous, resulting in further bone destruction. Extremely careful rehabilitation should be the rule. Finally the patient may progress to bespoke footwear with moulded insoles.

The rocker bottom Charcot foot with plantar bony prominence is a site of very high pressure. Regular reduction of callus can prevent ulceration. It ulceration does occur, an exostectomy may be needed. The most serious complication of Charcot foot is instability of the hind foot and ankle joint. This can leads to flail ankle on which it is impossible to walk. Reconstructive surgery and arthrodesis with a long-term ankle foot orlhosis has resulted in high levels and limb salvage ***(Papa et al, 1993).***

A second type of deformity is marked, ***pronated deformity,*** that resulting from medial displacement of the talonavicular joint or laterolantar calcaneocuboid dislocation.

Both deformities predispose ulcer formation in the midfoot. The early reorganization of osteo-arthropathy by assessing increased foot temperature (by

hand or thermometer), followed by prompt X-rays, is vital in early diagnosis and treatment. The design of custom-molded, supportive footwear with deeply molded insoles is needed to accommodate deformities after healing.

Joint Hypo mobility:

The relationship of joint limitation and plantar ulceration was established in a study by ***Delbridge et. al.*** *Significant joint limitation at the subtalar joint was found in diabetics with a history of ulceration compared to diabetics without ulceration and normal controls.* There was a significant correlation between joint mobility at the subtalar joint and mobility at the first metatarsophalangeal joint. Additionally, ***Mueller et al.*** found significant decreases in sensation, ankle dorsiflexion and subtalar joint motion in diabetic patients with ulceration compared to normal controls. They demonstrated the linkage of neuropathy and joint limitation with plantar ulceration in patients with diabetes.

Birke et al. demonstrated the relationship of hallux limitus with great toe ulceration. They found significantly decreased great toe extension using a torque range-of-motion system in diabetic patients with a history of great toe ulcers compared to diabetic patients with a history of ulcers at other sites and normal controls. ***Fernando DJS et al.*** studied the role of limited joint mobility (LJM) in causing abnormal foot pressures and foot ulceration. They found significantly increased foot pressures using pedobarography in diabetic patients with limited subtalar and metatarsophalangeal joints compared to diabetic patients and controls without limited mobility. Sixty-five percent of patients with neuropathy and limited joint mobility had a history of ulceration.

There was a strong correlation between plantar pressures and joint mobility. As shown by these studies, sensory loss and joint hypomobility may

result in increased pressure and plantar ulceration. Orthoses and footwear, designed to spread the stresses over time or reduce the function motion requirements (e.g., rocker sole) during walking time, are needed to compensate for hypomobility in the feet of patients with hypo mobility.

Connective Tissue Changes:

There is evidence that both the function and structure of proteins in diabetics are changed as a result of hyperglycemia. *Free glucose spontaneously attaches to proteins by a process called "nonenzymatic glycosylation".* Several investigations have shown that joint limitation may result from increased nonenzymatic glycosylation which leads to the molecular cross-linking of collagen protein and causes thickening and stiffness of periarticular tissues. ***Delbridge et al.*** observed similar increased nonenzymatic glycosylation of keratin protein in the stratum corneum of skin in 30 diabetic patients and proposed these abnormalities may contribute to hyperkeratosis and plantar ulceration .Repetitive stresses of gait are the primary cause of callus formation. Mechanical injuries develop from neglected, thickened callus that increases local pressure.

Thickness of the sole pad also may contribute to ulcer formation in diabetic patients. ***Gooding et al.*** found by sonography that thickness of the pad under the heel and first and second metatarsals was decreased in diabetics compared to controls. These decreases may be due to atrophy of muscle or connective tissue, or anterior migration of the metatarsal head pads associated with claw toe deformities.

2. Angiopathy

In patients with diabetic angiopathy until today, no histological or histochemical evidence has been found to define a specific type of diabetic arteriopathy. Consequently, diabetic arteriosclerosis is considered as a more

serious form of atherosclerosis characterized by its premature onset. *Hyperglycemia is assumed to be the crucial pathophysiological cause of the development of macroangiopathy and microangiopathy in diabetes mellitus.*

Apparently, hyperglycemia has a direct toxic influence on the arterial wall by increased accumulation of irreversible glycosylation end products, and secondly, it provokes endothelial dysfunction. The frequently occurring ulcerations of the diabetic foot are primarily caused by neuropathy; however, peripheral vascular disease (PVD) is often associated. *The risk of suffering from PVD in diabetic patients is approximately four-fold.* Usually, the distal segments of the lower leg arteries are concerned, where reconstructive intervention is complicated or even impossible. *Until now, there is no evidence for an association between an optimal control of blood glucose levels and a decrease in the risk of coronary heart disease, stroke, or PVD. In contrast, an attenuation of microvascular lesions is achieved by stringent control of blood glucose levels.* Thus, although the development of macroangiopathy may not be significantly influenced, the conduction of a strict control regimen of plasmatic glucose levels is advisable.

A. Macroangiopathy:

Diabetics have a higher incidence and prevalence of large vessel disease. Large vessel disease (atherosclerosis) may be present in the lower extremities and result in skin atrophy, hair loss, and coldness of the toes, nail dystrophy, pallor upon elevation, and mottling on dependence. *Macrovascular disease refers to changes in the medium to large-size blood vessels. The blood vessel walls thicken and become hard and non-elastic (arteriosclerosis).Blood vessels also become clogged with mounds of plaque (atherosclerosis). Eventually, the flow of blood may be blocked.*

Peripheral vascular disease refers to diseased blood vessels that supply the legs and feet. If blood flow is only partially interrupted, cramps, weakness, or pain in the legs when walking (claudication) may result. A completely blocked artery

will cause severe pain and the leg will become cold and pale. Treatments include replacing the diseased artery surgically or opening the blood vessel by compressing plaque against the artery wall (angioplasty).

Macroangiopathy (Atherosclerosis) is accelerated in diabetic patients compared to nondiabetic. ***It commonly involves the tibial and peroneal arteries.*** Atherosclerosis may result in foot ischemia characterized by intermittent claudication, pain with rest and elevation, ulceration and gangrene. The concomitant presence of neuropathy and ischemia predisposes the foot to minor trauma, which is often the precipitating ulceration and wound healing failure was the most common causal pathway to amputation. ***Ischemia was recognized in 46percent, neuropathy in 61 percent and infection in 59 percent of the cases. Loss of protective sensation was found in 82 percent. The importance of the minor trauma, skin ulceration and faulty wound healing is (72 percent).***

Early use of patient education and protective footwear for patients with loss of protective sensation could prevent this pathway. Noninvasive measurement of systolic blood pressures using Doppler ultrasound and digital photoplethysmography has been used to predict foot ulcers' healing potential. ***Wagner recommended pressure relief as the basic approach for treating foot ulcers when the ischemic index (ankle systolic pressure/ arm systolic pressure) is greater than .45*** . He reported a 90 percent healing rate for foot ulcers when this criterion was applied. Other criteria predictive of ulcer healing, using the ischemic index or systolic pressure, have been reported. ***Barnes et al. reported toe systolic pressure measurements to be more accurate than ankle systolic pressures in predicting wound healing in diabetic patients. They found 25-mm Hg toe pressure to be the lower limit for healing in the foot.***

Peripheral arterial disease is observed more frequently in diabetic compared with matched nondiabetic patients.

When ultrasound Doppler assessment of pulses is used, peripheral artery disease is found in 30% of all diabetic patients , the prevalence being 15 to 20% in those 70 yr of age and increasing to more than 50% at age 80 and above.

Lower limb atherosclerosis in the diabetic patient is characterized by preferential involvement of the lower leg, e.g., in 70% of diabetic patients compared with only 20% of nondiabetic patients who suffer from peripheral arterial disease. *Involvement of the deep femoral artery is also typical of diabetes.*

Often, multiple arterial segments are stenosed. The majority of patients do not complain of claudication. The risk factors predisposing to macroangiopathy and ischemic foot lesions are the same as the risk factors in nondiabetic patients, particularly smoking, but the diabetic patient has an excess risk for any given constellation of risk factors. *Peripheral arterial disease is 2.5 to 6 times more frequent than in nondiabetic patients and occurs 10 yr earlier.*

Calcification of the media of arteries is common in the diabetic patient and can be recognized by linear calcification on ultrasonography. Such calcification of the media is a risk predictor for severe atherosclerotic lesions of the intima. Media calcification makes measurements of arterial blood pressure unreliable and causes spuriously elevated values because of the reduced compressibility of the arteries.

B. <u>Microangiopathy:</u>

Diabetic microangiopathy is characterized by thickening of the capillary basement membrane, which may be caused by non-enzymatic glycocylation of collagen. These changes may result in transcapillary leakage of large protein molecules, such as albumin, and limited white blood cell movement. Decreased migration of lymphocytes would reduce resistance to infection.

Microvascular pathology has also been assumed to play a role in diabetic

neuropathy, and in the so-called diabetic foot.

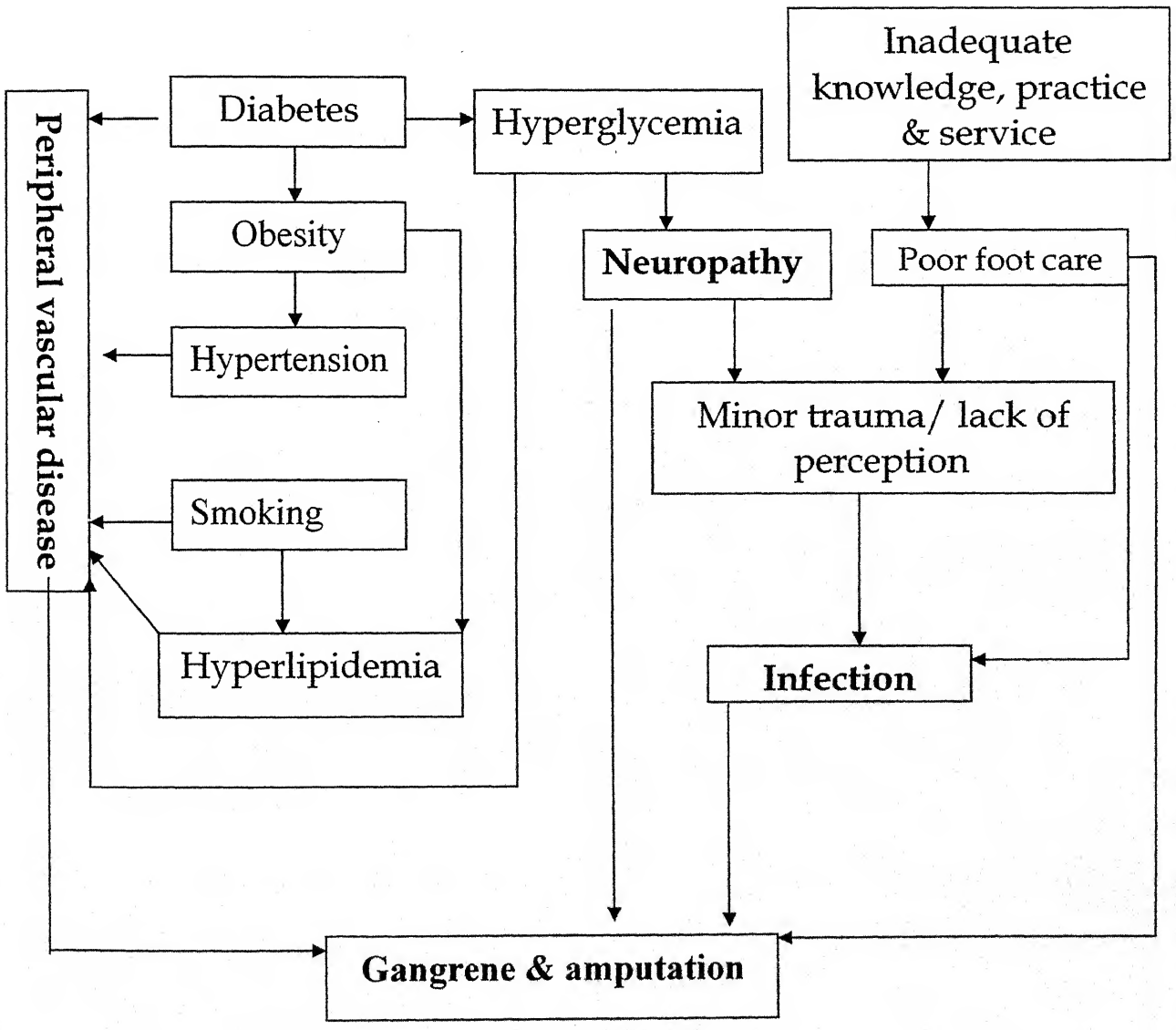
The histology of affected diabetic tissue reveals a PAS positive, thickened capillary basement membrane. Electron microscopy of skeletal muscle capillaries reveals reduplication of the basal lamina. The skin has not been thought to be a good sample source in evaluation of patients microangiopathy because small blood vessels of the dermis develop less basal lamina thickening than is found in skeletal muscle (which is also easily accessed using a needle biopsy).The structural changes which occur in the microcirculation do not seem to account for all the full extent of the disease, leading to the concept *of functional microangiopathy*. Some patients with severe microcirculatory problems such as gangrene of the foot have normal capillaries on skin or skeletal muscle biopsy. *Sluggish microcirculation resulting in micro-venular dilatation is considered "functional" in that it may be reversed with improved control of diabetes.* The clinical manifestations associated with this include retinal venous dilatation, red face, and periungual telangiectasia, all of which may be very early manifestations of the disease and which may improve with control of diabetes.

Functional microangiopathy may result from nonenzymatic glycosylation which affects many blood components including hemoglobin, red blood cell membrane, fibronectin, fibrinogen, and platelets. *Glycosylation of the red blood cell has been shown to inhibit the cell pliability and to decrease the ability of this cell to pass through pores smaller than 7 microns.* The lumen of some capillaries may be as narrow as 3 microns and ordinarily red blood cells will elongate into a more sausage like configuration to traverse these loops. Stiffened membranes will certainly inhibit or limit this passage. In addition to stiffened red blood cells, diabetics also have increased plasma concentration of fibrinogen and capillary leakage leading to loss of albumin and water. There is an increased tendency for

diabetic platelets to aggregate. The end result is increased whole blood or plasma viscosity and sluggish microcirculation.

In summary, it appears that microangiopathy can be attributed to both structural and functional abnormalities in these vessels.

SCHEMATIC DIAGRAM RELATING TO PATHOGENESIS OF P.V.D., NEUROPATHY, FOOT LESION & LIMB AMPUTATION



CLASSIFICATION OF DIABETIC FOOT

I. MODIFIED WEGNER'S CLASSIFICATION :

(Diabetic foot classification D.Vega et. al. Source control)

Grade	CLINICAL FEATURES
0	Normal Foot, variable degree of neuropathy, Joint deformities, Foot at risk.
1	Superficial ulcer, non affecting soft tissue Cellulites.
2	Non complicated deep ulcer, producing osteitis
3	Complicated deep ulcer, including deep infection I osteomyelitis and abscess.
4	Limited necrotizing gangrene (digital, plantar, heel).
5	extended gangrene

2. University of Texas Diabetic wound classification System.

Grade	Clinical Features
0	A pre or post-ulcerative site that has healed
1	Superficial wounds through the epidermis. or epidermis & dermis, that did not penetrate to tendon, capsule or bone.
2	Wounds that penetrate to tendon or capsule
3	Wounds that penetrate to bone or into joint

According to this classification system, within each wound grade, there are Four stages:-

- A. clean wound
- B. non ischemic infected wound
- C. ischemic non infected wound
- D. ischemic infected wound

3. Classification based on Diabetic Foot Surgery:-

Class	Procedure
I	Elective diabetic foot surgery (procedures performed to treat a painful deformity in a patient without loss of protective Sensation).
II	Prophylactic (procedure performed to reduce risk of ulceration Or re-ulceration in person with loss of protective sensation but Without open wound).
III	Curatives (procedures performed to assist in healing open wounds)
IV	Emergent (procedures lo limit progression of acute infection).

4. TREATMENT BASED DIABETIC FOOT INDEX:-

CATEGORY	FEATURES
0	Diabetic patients with intact protective sensation, good vascular status, and no previous history of ulcerations in the lower limb (minimal pathology present).
1	Patients with an insensate foot or diminish / loss of protective sensation, no previous history of ulceration in lower limb or charcot joints, no evidence of foot deformity
2	Neuropathic patients (insensate loot) with apparent foot deformity, no precious history of Neuropathic ulceration or charcot joints.
3	Similar to cat.2 but there is history of ulceration or charcot joint
4	Patients with an insensate injury i.e. Neuropathic ulcer or an acute charcot' joint.
5	Infected Diabetic foot, protective sensation major may not be *intact*. Charcot's joint may be evident.
6	Severely compromised vascular status (absent pedal pulses, monophasic Doppler sound, cold feet, absence of hair), Protective sensation major may not be present, A non-infected ulcer may or my not be present.

5.PendSey Classification {pendsey 2000}

1 - Neuropathic foot (in which neuropathy dominates).

2. Neuroischemic foot (in which vascular disease dominated)

A third type is also described in Indians:

3. Non-Neuroischemic foot- **Infected Foot** (in which infection dominates in absence of significant neuropathy or vascular disease).

NEUROPATHIC FOOT:-

This **warm, well perfused foot** with sensory deficit and autonomic dysfunction leading to arterio-venous-shunting and distended dorsal veins.

Peripheral autosympathectomy damage the neurogenic control mechanism, which regulates capillary and arterio-venous shunt flow. With loss of precapillary vasoconstriction, the skin may be dry and prone to fissuring.

Motor neuropathy also plays a role with paralysis of small muscles contributing to structural deformities, such as high arch and claw toes. This leads to prominence of metatarsal heads.

It has two main complications-

1. Neuropathic ulcer.
2. Neuropathic joint.

NEUROISCHEMIC FOOT:-

This is a **cool pulse-less foot** with poor perfusion. It also has Neuropathic ischemia resulting form atherosclerosis of the leg vessels.

This is often bilateral, multi-segmental and distal involving arteries below the knee. Intermittent claudication and rest pain, may be absent because of coexisting neuropathy.

Leg ulcers in neuro-ischemic foot develop on margins of the foot at sites made vulnerable by underlying ischemia to the moderate but continuous

pressure often from poorly fitting shoes.

In Indian Neuropathic foot is commonest accounting for 86.02% of case; non-Neuroischemic foot is responsible for 12.02% of case. Non Neuroischemic foot is responsible for only a minority of cases and account for 1.96% cases. These patients of Non-Neuroischemic foot are usually freshly detected cases of type 2 diabetes mellitus in which trivial trauma to foot leads infection in absence of significant neuropathy or vascular disease.

Undetected or uncontrolled diabetes is the culprit & on a few occasions limbs have been lost within a month of diagnosis of type 2 diabetes mellitus.

INFECTED FOOT:-

Infected foot is hallmark of Indian patients. Major factors complicating the issue are -

- Bare-foot walking.
-Lack of education.
-Surgical attempts at home.
-Use of unsterile topical application.
-Ignorance of primary care physician who often give resistant and small incision.
-Late reporting to the education centre.

By the time patient reports to a specialized centre, infection is already deep seated.

Infection of the plantar space accounts for majority of diabetic foot lesions. Continued ambulation in absence of pain due to neuropathy leads to further necrosis and spread of infection.

Such infected foot leads to complications due to compression of neurovascular bundles leading to further necrosis of distal foot and toes, or it

produces toxic thrombosis of the vessels leading to ischemia thus aggravating the existing problem.

The foot is often massively infected, foul smelling, and patient is toxic with signs of septicemia. Such an infected foot is limb threatening requiring urgent amputation and is at times even life threatening.

PERIPHERAL VASCULAR DISEASE (P.V.D.) AND DIABETES
(SAM & MOSES, 2000)

PVD causing arterial insufficiency is the most important factor related to the outcome of diabetic patient. Atherosclerosis and medial sclerosis are the most common arterial diseases.

Atherosclerosis causes ischemia by arterial narrowing and blockade. Medial sclerosis (Monckeberg sclerosis) is calcification of tunica media without involvement of arterial lumen. It produces a rigid conduct which severely interferes with indirect measurement of arterial blood pressure.

Micro angiopathy only should not be accepted as the primary cause of skin lesions.

Characteristic of atherosclerosis in diabetic patients as opposed to non diabetic patients.

*More common

*Affects younger individuals

*No sex difference

*Rapid progression

*Multi-segmental

*Prefers distal extremities.

Why diabetic patients are more prone to develop atherosclerosis is still unclear, but it is assumed that changes in circulating lipids results in a more atherogenic lipid profile with low HDL cholesterol and elevated triglyceride.

Within the diabetic population, nephropathy is the marker for generalized vascular disease and it is likely but not proven that these patients are more prone to develop PVD.

SYMPTOMS OF PERIPHERAL VASCULAR DISEASE (Fontaine)

Stage I: Occlusive arterial disease without clinical symptoms.

Stage II: Intermittent claudication.

Stage III: ischemic rest pain.

Stage IV: Ulceration / Gangrene.

This classification is imprecise in diabetic patients as these patients can have severe peripheral ischemia without symptoms. This is believed to be a consequence of loss of sensation due to peripheral neuropathy.

CLINICAL EXAMINATION

Vascular status in diabetic patients should be examined on an annual basis paying particular attention to-

1. History of intermittent claudication or ischemic rest pain.
2. Palpation of ***posterior tibial*** and ***dorsalis pedis*** pulses is mandatory.

When pedal pulses are absent ankle blood pressure should be measured with hand held ultrasound Doppler device. An ankle brachial pressure index [ABI= systolic ankle blood pressure divided by systolic arm blood pressure, both measured with the patient in supine position] below 0.9 indicates arterial occlusive disease.

3. Potential signs of critical ischemia:

-Blanching of feet on elevation.

-Dependent rubor, ulceration, skin necrosis or gangrene

However in diabetics due to peripheral neuropathy critically ischemic foot is relatively warm, with little discoloration.

CHRONIC CRITICAL ISCHEMIA

Critical ischemia is an indication of amputation of a major part of a limb, unless reversed by a revascularization procedure. It is defines by either of two following criteria -

1. Persistent ischemic rest pain requiring regular analgesia for more than two weeks.
2. Ulceration or gangrene of foot or toes both associated with ankle blood pressure of <50mmHg or Low systolic pressure of <30mmHg.

These are based on assumption that there are no differences between diabetic & non-diabetics concerning critical ischemia.

Factors contributing to abnormal foot pressure and possibly shear stress:-

Intrinsic factors	*Extrinsic factors*
Bony prominences	inappropriate foot wear
Limited joint mobility	Walking bare foot
Joint deformity	fall and accidents
Callus	Objects inside shows
Altered tissue properties	Activity level
Previous foot surgery	
Neuro-osteo-athropathic joints	

Diabetic foot wounds

- First phase of wound healing.

  -Inflammation: - Needs protection to prevent spread of infection.

- Second phase of wound healing.

  Proliferative Phase: - * Formation of fibro vascular granulation tissue followed by epithelisation.
  *Require proper moist environments
  *Difficult to infect

- Third phase wound healing (Phase of remodeling)

*Scar maturation

*Prevention of re-injury important

MULTIDISCIPLINARY DIABETIC FOOT UNIT

Management of diabetic foot requires the interaction of many medical disciplines. A team approach is needed that will save the limb not amputate.

Team members involved :

* Diabetiologist
* Vascular surgeon
* Neurologist
* Orthopedist
* Psychiatrist
* Pedorthist
* Infectious disease specialist
* Physical therapist
* Occupational therapist
* Social worker
* Home care nurse.

Major amputation can be avoided in about 88% of patients with limb threatening ischemia and in about 95% with foot ulceration complicated with infection. Multi-factorial treatment of complex foot lesions by a multidisciplinary foot care team is considered mandatory to obtain satisfactory salvage.

Screening technique to identify people at high risk for diabetic foot ulceration :

[Haupha M et.al., 2000]

Technique:-

A. Neuropathic symptom score.

B.Neuropathy disability score

C. Vibration perception threshold

D. Semmes Weinstein Monofilament (SWFs)

E. Peak planter foot pressure

F. Micro vascular oxygen measurements.

A. Neuropathy Symptoms Score (Boulton)

Patients were questioned about the presence or absence and possible nocturnal exacerbation of muscular cramps, burning pain, aching pain and irritation from bed clothes in the legs and feet.

Score 0 : Patient did not have a given symptom

Score 1 : Symptom (+)

Score 2 : Nocturnal exacerbation

Score 3 : Abnormal

B.Neuropathy disability score:

- Quantity the severity of neuropathy obtained from Physical examination.

- Based on examination of tendon reflex and sensory modalities.

***Reflex text:**

Score 0 : Patella and Achilles tendon, reflex-Normal

Score 1 : Reflex present with reinforcement

Score 2 : Reflex absent

***Sensory test:**

It includes a pinprick with a pointed metal or wooden pin, light touch with a strip of cotton ball, vibration with a tuning fork and temperature perception with a test tube filled with cold water. A score was given according to the anatomical location in which the patient could not identify the stimuli introduced.

Score 1 : Fail to perceive stimulus at the base of toe.

Score 2 : Failed to perceive the sensory stimulus at the mid foot.

Score 3 : Failed to perceive at the heal

Score 4 : Failed to perceive at the lower leg

Score 5 : Failed to perceive the stimulus at the knee.

The summation of reflex and sensory score for each modality was entered as the Neuropathic Disability Score (NDS).An NDS>5 was indicative of the existence of moderate to severe neuropathy.

C.Vibration perception threshold

It is measured by biothesiometer. This is a hand held device with a rubber tracer that vibrates at l00 Hz. The hand held unit is connected by an electrical cord to a base unit. This unit contains a linear scale that displays the applied voltage which ranges from 0-50 v. If a patient could not detect vibration at the maximum voltage of 50v, then a value of 51V were used. ***A value of 25 V was considered to be indicative of a patient at risk for foot ulceration.***

D.SemmesWeinstein Monofilaments (SWFs)

There is a set of 8 SWFs that apply pressure from 1 to 100 gm, to evaluate the cutaneous perception threshold. The plantar aspect of the hallux was used. With the eye closed the patient related to the investigator when he or she could feel the filament.

Inability to feel a 5.07WF (l0gm of pressure) was considered to be indicated of being at high risk foot ulceration.

E. Maximal Plantar Foot Pressure

The F-scan system is used to measure the dynamic plantar foot pressure.

The mat was calibrated for each patient by using the patient's weight before each testing session. The patient walks without shoes over mat and the maximal plantar foot pressure for the entire foot is obtained.

F. Micro-vascular Oxygen Measurement

Serial micro-vascular oxygen measurements may be used to identify at an early those ulcers that are unlikely to heal and therefore need surgical intervention.

Transcutaneous oxygen tension [TiO2] of 4.0kPa or more is essential for complete healing *(satyan M. Raj Bhadari et al, 1999).*

THE NATURAL HISTORY OF DIABETIC FOOT (EDMOSN ME et al., 2000)

The natural history of diabetic foot can be divided into six stages:

1. *The foot is normal and not at risk*. The patient does not have the risk factor that renders him vulnerable to the foot ulcer. These are neuropathy, ischemia, deformity, callus and edema.
2. *High risk Foot:* The patient has developed one or more of the risk factors for ulceration of the foot.
3. *Foot with ulcer:* Ulceration is on the plantar surface in the Neuropathic foot and on the margin in the neuro-ischemic foot.
4. *Foot with Cellulites:* The ulcer has developed infection with the presence of cellulites which can complicate both the Neuropathic and Neuroischemic foot.
5. *Foot with necrosis:* In the Neuropathic foot, infection is usually the cause. In the Neuroischemic foot infection is still the most common reason although sever ischemia can directly lead to necrosis.
6. The foot cannot be saved and will need a major amputation.

Every diabetic patient can be placed into one of these stages and the

appropriate management then carried out. In stage 1 and 2 the emphasis is on prevention of ulceration.

INVESTIGATIONS TO BE DONE IN A PATIENT OF DIABETIC FOOT

I) GENERAL:

Hb, TLC, DLC, Blood urea, creatinine, S. sodium, S. potassium, Random Blood sugar, fasting & post parandial blood sugar, Urine sugar and ketones.

Hemoglobin AIC: The patients on intensive insulin therapy, who keep accurate records of capillary blood glucose level, can easily monitor the adequacy of control of their disease and detect changing trends. **Hb_{AIC}** is an electrophoretically fast moving haemoglobin component that is present in normal persons & increase in amount in presence of hyperglycemia.

When properly assayed, the level of glycated haemoglobin gives an estimate of diabetic control for the preceding 3 month period.

Non-diabetic subjects, have Hb_{A1C} <6%, while level in patients with poorly controlled diabetes may have considerable above 10% (***Harrison's Textbook of internal Medicine, 15$_{th}$ed.)***

2) *LOCAL:-*

I] **X ray**: of foot area to rule out -

*foreign body

*osteomyelitis

* Subcutaneous gas

* Asymptomatic fractures

- Plain radiographs should be the initial imaging study in diabetic patients with signs and symptoms of a diabetic foot disorder. X-ray findings in a diabetic foot infection, such as osteomyelitis, may not demonstrate any osseous changes on radiographs for up to 14 days. Plain radiographs may

be indicated in the detection of osteomyelitis, osteolysis, fractures, dislocations seen in neuropathic arthropathy, medial arterial calcification, and soft-tissue gas.

ii] **Ultrasonogaphy**: USG is a widely available effective method that has not yet been used for the diagnosis of bone infection in diabetic foot. ***[Markus D. Enderl et al., 1999]***. A high resolution USG device may be used.

The findings are echoless zone with a distance of > 2mm adjacent to cortex of bone (elevation of periosteum) histopathologically confirmed as subperiosteal abscess.

In most sever cases; one can see a discontinuity of the cortex or an in homogenous echogenicity in projection of the compacta or spongiosa, representing bacterial sequesters.

In one study USG revealed 12 to 14 patients with osteomyelitis and was superior to X-ray in both detecting osteomyelitis and discriminating between the presence or absence of osteomyelitis. Soft tissue infection could be detected in almost all cases.

USG might have a better diagnostic power for detecting chronic Osteomyelitis in the diabetic foot than X-ray and has similar sensitivity and specificity as bone scan.

(iii)**CT SCAN/ MRI:**

CT SCAN: Computed tomography (CT) scans may be indicated in the assessment of suspected bone and joint pathology not evident on plain radiographs. This study offers high anatomic detail and resolution of bone with osseous fragmentation and joint subluxation being well visualized.

MRI SCAN: It is the most superior non-invasive imaging method for the diagnosis of osteomyelitis.

MRI should be added to the diagnostic procedure if at first USG dose not

shows osteomyelitis despite high clinical suspicion or if any surgical therapy besides minor debridement is, indicated ***(Markus D. Enderl et. al.1999).***

Magnetic resonance imaging (MRI) is often used in evaluating soft-tissue and bone pathologies. This scan may be indicated to aid in the diagnosis of osteomyelitis, deep abscess, septic joint, and tendon rupture. It is a readily available modality which has a very high sensitivity for bone infection and can also be used for surgical planning. Despite its high cost, magnetic resonance imaging has gained wide acceptance in the management of patients with diabetic foot infections.

(iv)**Bone Scan**: Technetium bone scan is sensitive but expensive and lacks adequate specificity for diagnosis of osteomyelitis. ***Indium labeled leukocyte scanning is considered to be the most accurate radio nucleotide study***. But it is too expensive & it may be difficult to interpret in presence of local soft tissue inflammation ***{Markus D. Enderl et al. 1999}.***

Technetium bone scans are often used in diabetic foot infections although this modality lacks specificity, especially in the neuropathic patient. Three-phase bone scans may be indicated in the early detection of osseous pathology such as osteomyelitis, fractures, and Charcot arthropathy. However, such imaging tests are best utilized to confirm clinical suspicion and have higher specificity when combined with other scintigraphic techniques such as white blood cell scans.

Gallium 67 citrate is another nuclear medicine technique that is not used as frequently today due to more accurate alternative imaging studies. This study can be used in concert with technetium bone scans to aid in the diagnosis of osteomyelitis and also may be of value in the presence of acute osteoarthropathy.

Indium-111 leukocyte scans, Tc_{GG}-labeled white-cell scan (HMPO), or other variations of white blood cell scintigraphy are useful in differentiating between osteomyelitis and neuropathic arthropathy due to their relatively high sensitivity

and specificity. These tests are expensive and time consuming, but are available at most hospitals when early identification of bone infection is required.

3} HISTOPATHOLOGY *(MARVIN E. LEVIN, 1993)*

Histopathology should be considered when ulcer appears at atypical location e.g. not over the metatarsal heads or plantar surface of the hallux, when it cannot be explained by trauma and when it is unresponsive to aggressive therapy. On numerous occasions biopsies of a typical ulcer have revealed malignancies, both primary and metastatic.

4} **Culture:**

Clinically uninfected ulcer should not be cultured. Superficial swabs from an infected ulcer are not ideal for culturing since both colonizing and infecting organisms are recovered. Infecting organisms are more reliably detected in specimens obtained by curettage of base of ulcer after debridement ***[Lipsky BA et al, 1990).*** Needle aspiration is a reliable method of detection but its sensitivity is low ***[Wheat j et. al.; 1935-40].***

Culture of bone specimen is obtained by percutaneous biopsy or surgical excision [through approaches that do not traverse the ulcer] is the best method of determining the cause of osteomyelitis ***[Gregory M caputo et. al.].***

Causative organism

Most mild infection is caused by aerobic gram positive cocci [Staph aureous & strep pyogens] ***{John EW et al.1984}.***

Deeper life threatening infections is caused by polymicrobial & aerobic gram positive cocci, gram negative bacilli [E. coli, Klebsiella, and Proteous] & anaerobes [bacteroids & peptostreptococcus] ***{Wheat j et. al. 1935-40}***

5. NON INVASIVE VASCULAR LABORATORY TESTS

When the history and physical examination suggest ischemia or the presence of a nonhealing ulcer with absent pedal pulses, further noninvasive testing is warranted. Noninvasive arterial studies (NIAS) should be performed to

determine lower extremity perfusion. Such studies may include Doppler segmental arterial pressures, and waveform analysis, ankle-brachial indices (ABI), toe pressures, and transcutaneous oxygen tension ($TcPO_2$).

Vascular consultation should be considered in the presence of abnormal noninvasive arterial studies and a nonhealing ulceration. Arteriography with clearly visualized distal runoff allows appropriate assessment for potential revascularization. Digital subtraction angiography (DSA) or magnetic resonance angiography (MRA) are alternatives for evaluation of distal arterial perfusion.

I. **Doppler Ultrasound:** a continuous wave ultrasound signal is beamed at an artery & the reflected beam is picked up by a receiver. The change in the frequency of the reflected beam is compared with that of the transmitted beam is due to Doppler shift, resulting from the reflection of the beam by moving cells. The frequency change may be converted into an audio signal & in pulsatile sound typically results. *Doppler ultrasound equipment can, therefore, be used as a very sensitive type of stethoscope in conjunction with a sphygmomanometer to assess the systolic pressure in relatively small vessels; this is often possible even at sites where arterial pulses can not be palpated.*

The ankle brachial pressure index [ABPI]: can be easily assessed. Generally, the higher of the recordings of pressure in dorsalis pedis and posterior tibial arteries serves as numerator, with the high systolic pressure between brachials serving as a denominator. The resting ABPI is normally about 1.0, values below 0.9 indicates some degree of arterial obstruction. A value less than 0.3 suggest imminent necrosis.

A Doppler ultrasound probe can also be used to assess differences in arterial blood pressure between segments of a limb, there by giving an indication of site of stenosis. In leg the cuff is commonly placed above the ankle, at mid calf and mid thigh to provide segmental pressure. Artifacts are due

especially to calcified arteries, which may be incompressible and lead to a falsely high limb pressure or ABPI. This is particularly the cases in diabetics.

(ii)Duplex Imaging: This is an investigative technique of major importance in vascular disease. A duplex scanner uses B-mode ultrasound to provide images of vessels. These images are created through the different ability of different tissues to reflect the ultrasound beam. A second type of ultrasound, namely Doppler ultrasound, is then used to isolate the imaged vessels and the Doppler shift obtained is analyzed by a dedicated computer in the duplex scanner. Such shifts can give detailed knowledge of vessel blood flow, turbulence etc. some scanner have the added sophistication of color coding which allows visualization of blood flow on the image. The various colors indicate change in direction & velocity. ***It should be appreciated that modern duplex scan is at least as accurate as angiography in certain circumstances.*** It is preferred to angiography in terms of cost effectiveness & safety.

iii] **Plethysmography**: - It assesses changes in volume of a limb or digit over the cardiac cycle. Air filled cuffs or mercury in rubber strain gauges have typically been used. For most clinical purposes test has been superceded by duplex scanning.

iv] **Pedography:-** Pedography as a measurement of foot for early reorganization of altered pressure pattern in the individuals with diabetes.

In the early 1980's studies was first published looking at the changes in dynamic pressure distribution patterns of persons with diabetes.

Inspired by studies conducted in England with the pedobarograph, the first electronic pressure distribution measuring system, ***Kirsh, Scahff, & Setiz*** made their initial test in Germany [1983] to examine the pressure distribution changes in neuropathic foot with a new measurement technology.

Their new measurement system, known as e-med and developed by

Novel, was used in these studies. For the first lime, more precise data could be provided with the e-med capacitive sensor technology. The neuropathic foot tests clearly showed a difference when compared lo healthy feel.

In 1984 the Bundesministerium fuer Forschung and technology (State Department for Research and technology) sponsored a project led by professor Mehnert and conducted by Dr. Dieter Kirshc from the Munich Diabetes Research Group. The Research Group examined more than 600 persons.

The diabetics with neuropathy could easily be identified from their specific foot pressure patterns during dynamic loading.

It was documented that during the roll over process, the neuropathic fore foot displayed increase & longer loadings than the healthy forefoot. The neuropathic patient also exhibited a "slap" gait. The load on the heel and toes was lower than the unaffected feet.

Many neuropathic feet had substantially higher localized pressure, particularly at the metatarsal area, than the non-neuropathic feet.

With data analysis of the healthy control group it was quickly determined that the non-diabetic, due to various foot deformities, could record similar pressure pattern to the neuropathic diabetic. The frequency of the altered pressure pattern in the patients with diabetes was however substantially increased. Additional testing procedures, such as temperatures, vibration and monofilament were used as complimentary documentation.

The result was significant if the pressure distribution showed a typical change, the other monitoring tests recorded changes as well. The pressure distribution measurement procedure worked in conjunction with these tests for outcome documentation.

It has been theorized for some time that the areas of increased pressure on the diabetic foot are the areas of highest risk for tissue breakdown.

Consequently, it was suggested that a relationship between the absolute pressure and the risk for tissue breakdown could be established.

Currently there is no association localized pressure threshold, which guarantees tissue breakdown.

However, in current studies it is noted that 50 to 60 N/cm_2, measured on a calibrated e-med platform with 2 $sensors/cm_2$, can suggest risk threshold, the recorded localized maximum pressure is independent upon the sensor technology, specifically the sensor resolution and the precision in calibration of the sensors therefore, results from other research studies using pressure distribution technology, cannot be directly compared.

Review of the pressure picture gives the following basic information. The entire display of the maximum pressure picture allows for comparison of foot regions. It may be necessary to consider the duration of contact in addition to the absolute pressures. The Charcot foot displays higher pressures in the mid foot region as compared to the typical diabetic foot pattern. Pedography may aid early detection of deformed feet.

Naturally, the pressure picture of the neuropathic diabetic patient can be influenced by foot deformities and incorrect foot function. It any case, the specific foot regions with higher pressures indicate greater risk for tissue breakdown.

In addition to the standard therapy for diabetic patient, and altered load on the foot with localized high pressures may require, immediate foot treatment with appropriate footwear and pressure reliving orthotics. Under no circumstances should these patients walk barefoot because plantar pressures will increase dramatically and the foot is left unprotected. Shoes and orthotics should be fabricated by a trained professional. The 1:1 hardcopy of the Pedography platform measurement provides a baseline for the construction of

the shoe insert.

The patient can then be measured inside the modified shoe for quantification of the load. In shoe pressure distribution measurement systems were designed for that purpose.

The goal for the diabetic patient shoe insert is to create a uniform pressure to prevent localized high pressures. The patient should be instructed to examine their feet. During the patient visit, the color display and print out may be used to educate the patient about the areas of concern.

Pedography is a quick measurement tool and it does not place any burden on the individual's feet. This is precisely, why pressure assessment, so invaluable.

The objective for health professionals is to reduce ulceration and amputation in the diabetic population. Pedography is a cost effective method to help reach this goal.

The typical pressure pictures of Neuropathic feet show three factors:

1. No definite roll-over process from heel to mid foot to forefoot and finally to the toes. The foot is typically in a foot flat position at ground contact and is characterized by immediate forefoot loading. These patterns can also be recognizing from the centre of pressure line in the foot pressure picture.
2. The localized pressure values under the 3^{ed},4^{th} , 5^{th} metatarsal heads, are often elevated in relation to other foot regions. Pressure of $50N/cm_2$ may be seen with the e med 2 sensors/cm^2 platform and in some cases over $100N/cm^2$ recorded.
3. The toes may be less pronounces or not visible in the dynamic picture due to diminished function.

FOOT INK SCAN

An alternative method of obtaining pressure point picture is foot ink scan

which is very useful in our setting, which shows increased pressure over metatarsal heads and heel.

TREATMENT PLAN:

1) Metabolic control
2) Mechanical control
3) Wound Control
    (i) Antibiotics
    (ii) Dressing
    (iii) Aggressive Debridement
    (iv)Stimulation of the wound healing
4) Medical therapy for increasing vascularisation and decreasing oedema.
5) Patient's education
6) Modified 'BOOT' therapy
7) Offloading of pressure points
8) Special modified shoes based on pressure points.
9) Surgical therapy to improve circulation.

1. METABOLIC CONTROL

A. **Diet:** This depends upon type of diabetes. In IDDM, particularly those on intensive insulin regimens; the composition of the diet is not of critical importance since adjustment of insulin can cover wide variations in food ingestion. In NIDDM patients not treated with exogenous insulin, more rigorous adherence to diet is required, since the endogenous insulin reserve is limited. Such patients cannot respond to the increased demand produced by excess calories or increased intake of rapidly absorbed carbohydrate.

In Insulin requiring patients, the distribution of calories is also important if hypoglycemia is to be avoided. A typical pattern might include 20% of total for breakfast, 35% for lunch, 30% for dinner and 15% as a late evening feeding

(Harrion's Text Book of Medicine, 15 Ed.)

B.**Insulin:** Insulin is required for treatment of all patients with IDDM and many patients with NIDDM. If the physician does not use oral hypoglycemic agent, all diet unresponsive NIDDM subject, must be given the hormone. It is fairly easy to control the symptoms to maintain a normal blood sugar throughout 24hr even with the use of multiple injection of regular insulin of infusion pumps.

CONVENTIONAL PREPARATIONS OF INSULIN *[K.D.T., 5TH Ed]*

Type	Appearance	Onset (hrs.)	Peak (hrs.)	Duration (hr)	Can be mixed with
Short Acting					
Regular Insulin (Soluble)	Clear	0.5-1	2-4	6-8	All preparations.
Prompt Insulin zinc Suspension (amorphous) or semilente	Cloudy	1	3-6	12-16	Regular, lente preparations
Intermediate acting					
Insulin zinc Suspension or lente(ultra : Semi :: 7 : 3)	Cloudy	1-2	8-10	20-24	Regular. Semilente
Neutral protamine Hagedorm (NPH) or Isophane insulin	Cloudy	1-2	8-10	20-24	Regular
Long acting					
Extended insulin Zinc suspension(Crystalline) or Ultralente	Cloudy	4-6	14-18	24-36	Regular Semilente
Protamine zinc insulin (PZI)	Cloudy	4-6	14-20	24-36	Regular

Three treatment regimens are given ***{Harrison's text book of internal medicine 15th Ed.}***

1. Conventional
2. Multiples subcutaneous injections [M.S.I.]

3. Continuous subcutaneous insulin infusion (C.S.I.).

MSI and CSI are required in intensive treatment schedules designed to protect against complications.

I) **Conventional Insulin Therapy** involves the administration of one two injections a day of intermediate acting insulin with or without the addition of small amounts of regular insulin. Adults of normal weight may he started on 15-20 units per day. Obese patient because of insulin resistance may be started on 25 to 30 units a day. In a single dose schedule one might begin with 20 units of intermediate and 5 units of regular insulin.

ii) **Multiple subcutaneous insulin injection**. Most commonly involves administration of intermediate or long acting insulin in evening as a single dose together with regular insulin prior to each meal.

Adjusting Insulin Dosage in a Multiple- Injection Schedule

1. 0.6 to 0.7 unit's insulin per Kilogram body weight.
2. 25% NPH insulin at 9 p.m.; 75% regular insulin in divided doses (40% before breakfast, 30% before lunch, 30% before supper).
3. Adjust NPH insulin every 48h based on fasting blood glucose

<3.3 mmol/L (<60mg/dl) - 2 units

>5.0 mmol/L (140 m/dl) + 2 units.

4. Adjust regular insulin every 48h based on 1 -hr postprandial glucose.

<3.3 mmol/L [< 60 mg/dL] - 2 units

> 7.8 mmol/L [> 140 mg/dL] + 2 units

During the initiation of therapy, the insulin dosage is changed until the target range is reached. After initial stabilization, a variable insulin schedule is prescribed to maintain tight control. For example, if after initiation, the patient is found generally to require 12 units of regular insulin before breakfast but has a pre-breakfast blood sugar level of 8.9 mmol/L (160mg/L), 15 units of regular

insulin instead of the usual 12 would be taken. (*Source: Schiffrin and Belmonte).*

DAILY THERAPY

Pre-prandial glucose mmol/L	Pre-prandial glucose m.gm/dl	Regular insulin units
<3.3	<60	-2
3.4-5.0	61-90	no change
5.1-6.7	91-121	+1
6.8-8.3	121-150	+2
8.4-11.0	151-200	+3
11.1-13.9	201-251	+4
>13.9	>250	+6

Multiple subcutaneous insulin injection can be effective in controlling the plasma glucose level and in some studies it appears to match goals achieved with CSII.

iii) Continuous Subcutaneous Insulin Infusion: It involves the use of a small battery driven pump that delivers insulin subcutaneously into abdominal wall, usually through 27-G butterfly needle. With CSI -Insulin is delivered at a basal rate continuously throughout the day with increases in rate programmed prior to meals. Adjustments in dosage are made in response to measured capillary glucose. Ordinarily about 40% of total daily dose is given at a basal rate, the remainder being administered on pre-prandial boluses.

For surgical procedures in diabetic foot patients intermediate insulin is omitted and treatment is carried out with the regular

MORE PURIFIED INSULIN PREPARATIONS:-

The conventional or standard preparation of insulin contains 1%

(10,000 ppm) or more of other proteins (proinsulin, pancreatic proteins, insulin derivatives etc.) which are potentially antigenic.

In 1970 improved purification techniques were applied. This resulted in the availability of highly purified and practically non antigenic preparations.

Single species insulin from pork or beef has been made available. Pork insulin, being more homologous to human insulin is less immunogenic. These can then be modified like conventional preparations into large acting form according to the purification method used. The preparations can be categorized into:

1. Single peak insulins: Purified by gel filtration and repeated crystallization, they contain 50-200 ppm pro-insulin.

2. Monocomponent (MC) insulins: After gel filtration it is further Purified by ion-exchange chromatography; the content of Proinsulin is reduced to <20 ppm.

The immunogenicity of pork MC insulin is similar to that of human insulin, while the single peak preparations still have significant immunogenicity. These preparations are most expensive, but offer the advantage of greater stability, less allergic reactions, less insulin resistance and less lipodystrophy.

Human insulins: In the 1980 the human insulins were produced by recombinant DNA technology in Escherichia coli;

- Proinsulin recombinant bacterial (prb) and in yeast.
- Precursor yeast recombinant (pyr) or by enzymatic modification of procine insulin (emp).

In developed countries the use of human insulins has rapidly overtaken that of conventional and purified animal insulins. Human insulin is most water soluble as well as hydrophobic than procine or bovine insulin. It has a most

rapid S.C. absorption earlier and most defined peak and slightly shorter duration of action.

c) **Oral Agent**: NIDDM that cannot be controlled by dietary management often respond to sulfonylurea. The drugs are easy to use and appear to be safe, Nevertheless use of oral agents has decreased in response to the emphasis on better control as means of slowing the development of late complications. These drugs can bring plasma glucose back to normal in some patients with relatively mild crises, but in patients with significant hyperglycemia, plasma glucose tends to improve but not to approach the normal range in response to their agents. Thus a high percentage of patients with NIDDM neither are nor treated with insulin ***(Harrison Text Book of Medicine 15^{lh}Ed.)***

Oral Hypoglycemic agents

Agents	Daily Dose mg	Doses per clay	Duration of action (hr)	Clearance Route
Sulphonylureas				
1.chlorpropramide	100-500	1-2	36-48	K,L
2.Acetohexainide	500-1500	1-2	12-18	L
3.Tolbutamide	500-3000	2-3	6-8	L
4.Tolazamide	125-1000	1-2	18-24	L
5-Glibenclamide	5-15	1-2	18-24	L
6.Glipizidc	5-20	1-2	12-18	L
7.Glimepiride	1-4	1	12-24	L
Biguanides				
l.Phenoformin	25-150	1-3	8-12	L-K
2.Metformin	500-2000	2-4	6-8	K
Thiazolidinediones				
l.Troglitazone	400-600	1	Upto24	L

Two peptides are under evaluation as adjunct treatment for NIDDM. Both insulin-like growth factor 1 (GLP-1), a peptide derived from the

proglucagon molecule, lower blood glucose levels in normal subjects and in patients with diabetes, GLP-1 (7-36) and (7-37) amides are the lead agents under trial. Their usefulness is not established ***(Harrison's text Book of Medicine, 15th Ed.)***

Attempts are continuing to make alternative routes of insulin delivery (nasal, oral) practicable, Liposome insulin's is being tried. One such product ***'Lyspro insulin'*** is absorbed rapidly as a monomer and has shorter duration of action may offer greater flexibility in timing of insulin injection before meals and may reduce late postprandial hypoglycemic episodes.

2. MECHANICAL CONTROL:

Breaks in the Skin - Skin integrity is of utmost importance in the diabetic patient with neuropathy and/ or ischemia. A break in the skin, no matter how small, can permit the introduction of bacteria. Common sources of breaks in the skin are fissures, blisters, toenails trauma and corn / calluses.

Fissures are most common caused by dry skin. The most common location is along the edges of the heels, adjacent to the thickened nails, and on the medial part or edges of the big tow joint. Diabetic patients with peripheral neuropathy tend to develop cornified, thickened skin as a result of glycosylation of protein elements of the skin.

Ischemia will cause skin to become thinner & less resistant and cause skin, in turn, to become dry and prone to cracking.

Any chronic or acute dermatitis of the skin may permit fissures to develop fungal infections. Maceration of skin between toes because of perspiration or incomplete drying after bathing also can cause fissuring.

Blisters are cause most frequently by friction from shoes, a sudden increase in activity, wrinkles in socks, and foreign bodies in shoes. Heat, intense cold or chemical irritants may also cause blistering.

Toe nails undergo dystrophic changes with advancing age due to trauma, hereditary, circulatory changes, dry skin changes, mycotic infections & psoriasis. As the nails begin to change shape and thicken, the adjacent skin is at risk of breaking and becoming infected.

Corns and Calluses:-

CORN is a horny induration and thickening of the skin that may be hard or soft according to location. Pressure, friction, or both cause this condition.

CALLOSITY it is a circumscribed thickening and hypertrophy of the horny layer of the skin. It may be oval or elongated, gray or brown, slightly elevated, with a smooth burnished surface. It appears on the flexor surfaces of hands and feet and is caused by friction, pressure, or other irritation.

They arise over bony prominences & are normal response of skin to friction. Under normal circumstances, these lesions become painful as their thickness increases. The presence of neuropathy can mask the pain and allow a blister to develop under the lesion. The blister may break outside, become infected and lead to an ulceration, with potential abscess formation or osteomyelitis.

Deformity must be accommodated and callus, dry skin fissures and oedema must be treated.

DEFORMITIES

Deformities in the neuropathic foot tend to render the plantar surface vulnerable to ulcers, requiring special insoles whereas in the Neuroischemic foot, the margins need protection and appropriately wide shoes should therefore be advised.

Footwear can be divided into three broad types: ***sensible shoes*** (from high street shops) for patients with minimal sensory loss, ***ready made stock*** (off the shelf) for Neuroischemic feet that need protection along the margins of the foot

but that are not greatly deformed and ***customized or bespoke (made to measure) shoes*** containing cradled, cushioned insoles, these are necessary to redistribute the high pressures on the plantar surface of the neuropathic foot.

CALLUS

Patients should never cut their callus off or use callus removers. It should be removed regularly by sharp debridement.

DRY SKIN AND FISSURES

Dry skin should be treated with an emollient such as E 45 cream, calmurid cream or petroleum jelly.

OEDEMA

Oedema may complicate both the neuropathic and the Neuroischemic foot. Its main cause will be impairing cardiac and renal function, which should be treated accordingly. Oedema may rarely be secondary to neuropathy. It responds to Ephedrine starting at a dose at 10 mg t.d.s. and increasing up to 30-60 mg t.d.s.

2. WOUND CONTROL

(A) ANTIBIOTICS

The selection of an antibiotic or parenteral antibiotic for the treatment of a diabetic foot infection is based on medical judgment. The oral antibiotics that are should be effective for gram positive and gram negative organisms.

The criterion for hospitalization and treatment with parenteral antibiotics include patients, who are septic, febrile and have leukocytosis.

Indication of Worsening Infection

Not all neuropathic foot ulcers are infected. Infection is suggested by local inflammation, purulent drainage, sinus tract formation or crepitation. The severity of cellulites may range from a mild, localized infection to a limb threatening, necrotizing process with fascitis. Foot ulcer can be divided into *superficial* lesions that do not threaten the limb deeper, limb threatening ulcers.

Fever, chills, and leukocytosis are absent in two thirds of patients with limb-threatening infection, which may include deep abscesses, extensive soft tissue infections, or metastatic infection of remote sites ***(Gibbons GW et al., 1984).*** Hyperglycemia is a common sign of limb-or life-threatening infection. Erythema swelling and warmth in a non-ulcerated foot may indicate acute Charcot's disease rather than infection. Conversely, some un-inflamed ulcers are associated with underlying osteomyelitis ***(Newman LG et al, 1991).***

Sign and Symptom

- Increased
  - Drainage
  - Erythema
  - Pain
  - Temperature
- Malodorous
- Lymphangitis
- Lymphadenopathy
- Gangrene
- Laboratory :- Increased – Blood Sugar
  - W.B.C.
  - E.S.R.

SELECTED EMPIRICAL ANTIMICROBIAL REGIMES FOR FOOT INFECTIONS IN PTS WITH DIABETES (Gregory m.Caputo et al.)

Non Limb Threatening Infections	**Limb Threatening Infections**	**Life Threatening Infections**
Oral Regimen	**Oral Regimen**	**Parenteral Regimen**
Cephalexine	Fluroquinolones	Imipenum-Cilastin
Amoxicilline-clavurunate	Clindamycin	Vancomycin
Parenteral Regimen	***Parenteral Regimen***	
Cefazoline	Ampicilline-Sulbactum	Metronidazole
Oxacilline	Ticarcilline-clavurunate	Aztreonam
Nafcilline	Cefoxitin/cefotetan	Ampicilline-Sulbactum
Clindamycin	Fluroquinolones & Clindamycin	Aminoglycocides

The Optimal duration of antimicrobial therapy after surgical debridement of infected pedal bone has not been established. Traditionally, a regimen of four to six weeks of parenteral antibiotic therapy has been recommended; however, a two to three week course of antibiotics directed at residual soft tissue infection is reasonable. In contrast prolong therapy is recommended for tarsal or calcaneal osteomyelitis since the infected bone is debrided piecemeal.

(B) *DRESSING*

Sterile non adherent dressing should cover all ulcers to protect them from trauma, absolute exudates, reduce infection and promote healing. There is no evidence to support a particular dressing ***(Mason JM et al., 1999).*** The following dressing properties are essential for the diabetic foot-care and speed of shifting, ability to be walked on without disintegrating and good exudates control. Dressing should be lifted everyday to ensure that problem or complications are detected quickly, especially in patients who lack protective pain sensation.

Dry dressings are used in casts, otherwise, saline-moistened sterile gauze is applied and changed two to three times a day. Occlusive dressings also appear to be satisfactory ***(Hutchiason JJ et al, 1990).*** The efficacy of topically applied growth factors has not been conclusively proved ***(Steed D et al., 1992).*** *Topical iodine preparations, astringents and hydrogen peroxide interfere with the healing of the wound* **(Kucan Jo et al., 1981).**

Commonly used dressings material in diabetic foot

Solutions

* Dilute cetrimide
* 1% Providone iodine Oxofferin
* H_2O_2 - very rarely - should be condemned

Ointments & Creams:

Antibiotics *Gentamycin

*Silver sulphadiazine

*Mupirocin

*Sofradex for skin reactions

*Metrogyl (Metronidazole)

Enzyme ointment

*Collagenase (Salutyl)

Softening agents

- Plain petroleum jelly
- Moisterex
- Aloderm
- Coconut oil

Platelet derived growth factor

-Regranex

Hydrocolloid drugs

-Inteasite gel - hydrocolloid gel

-Duoderm - Karaya gum

-Kaltostat - Sea weed

-Medifil - collagen

-Opsite

-Tulle gauze - plain, antibiotic impregnated

-Inteasite gel - cutinova hydra

Occlusive dressing

Advantages: reduce pain, rapid healing, autolytic debridement, increased granulation, reduced friction.

Disadvantages: Maceration; Accumulation of pus; Adherence to healthy tissue, increased bacteria; May promote anaerobic growth.

(c) ***Aggressive Debridement:*** Debridement of necrotic tissue is an integral

component in the treatment of chronic wounds since they will not heal in the presence of nonviable tissue and debris. Adequate debridement must always precede the application of topical wound healing agents, dressings, or wound closure procedures. Types of debridement are autolytic, enzymatic, mechanical, and surgical.

Surgical debridement is a key component and a cornerstone in the management of diabetic foot ulcers. Thorough sharp debridement of all nonviable soft tissue and bone from the open wound is accomplished primarily with a scalpel, tissue nippers, and/or curettes. Excision of necrotic tissue extends as deeply and proximally as necessary until healthy, bleeding soft tissue and bone are encountered. Any callus tissue surrounding the ulcer must also be removed. A diabetic ulcer associated with a deep abscess requires hospital admission and immediate incision and drainage. Joint resection or partial amputation of the foot is needed in the presence of osteomyelitis, joint infection, or gangrene. Necrotic tissue removed on a regular basis can expedite the rate at which a wound heals and has been shown in a recent study to increase the probability of attaining full secondary closure. Less frequent surgical debridement can impact negatively on the rate of wound healing and secondarily increase the risk of infection. Surgical debridement is repeated as often as needed if new necrotic tissue continues to form. ***Weekly debridement is commonly required.***

Off-loading

Reducing pressure to the diabetic foot ulcer is an essential component of treatment. Without proper off-loading and pressure reduction, ulcers will continually be traumatized to the point that they cannot heal.

The choice of off-loading modality should be determined by the patient's physical characteristics and ability to comply with the treatment, as well as the location and severity of the ulcer. Various centers prefer specific initial modalities,

but the clinician frequently must alternate treatments based upon clinical progress of the wound. It is not unusual to practice step-up therapy where increasingly effective modalities are used when little improvement is noted with initial therapy. Some centers prefer to apply total contact casts (TCC) initially and then step-down to less restrictive modalities when lesions have healed or are nearly healed.

- The following off-loading techniques have been found to be useful in the management of diabetic foot ulcers:
- Total nonweightbearing: crutches, bed, wheel chair
- Total contact casting
- Foot casts or boots
- Removable walking braces with rocker bottom soles
- Total contact orthoses - custom walking braces
- Patellar tendon-bearing braces
- Half shoes or wedge shoes
- Healing sandal - surgical shoe with molded plastizote insole
- Accommodative dressings: felt, foam, felted-foam, etc.
- Shoe cutouts (toe box, medial, lateral, or dorsal pressure points)
- Assistive devices: crutches, walker, cane, etc.
- It is critically important to remove the patient from the shoes that caused the ulcer. In fact, the consensus of opinion is such that no patient with an active foot ulcer should be placed back into an unmodified shoe until complete healing has occurred.

Debridement reduces the bacterial load on the ulcer even in the absence of over infection, restores chronic wounds to acute wound, and releases growth factors to aid the healing process ***(Edmonds ME et al., 2000)***. It also enables a deep rural to be taken for culturing the larvae of the green bottle fly which are sometimes

used to debride ulcer especially in the neuroischaemic foot ***(Rayman A ct. al., 1998).***

In diabetic foot patient debridement should not be delayed. Diabetic patients do not tolerate undraled suppuration. Failure to debride necrotic infected tissue and drain purulent collections increases the risk of amputation. Drainage by needle aspiration must be performed independently of the status of the arterial circulation with revascularization postponed until sepsis is controlled***(Ernst SD et al)*** Adequate debridement may require multiple procedure.

(d) Stimulation of wound Healing

Techniques to stimulate wound healing include:

Regranex (Platelet derived growth factor)

Derma graft

Apli graft

Hyaff

Vacuum assisted closure

Regranex

Platelet derived growth factor (Regranex), stimulates fibroblasts and other connective tissue cells, locates in the skin and is beneficial in enhancing wound healing processes of cell growth and repair. It is applied once daily.

Dermagraft

Dermagraft is an artificial human dermis manufactured through the process of tissue engineering. Human fibroblast cells obtained from neonatal foreskin are cultivated on a three dimensional polyglactin scaffold. As fibroblasts proliferate within the scaffolds, they secrete human dermal collagen, growth factors and other proteins, embedding themselves in a self-produced dermal matrix. This results in metabolically active dermal tissue with the

structure of a papillary dermis of newborn skin (*Naughton G et al., 1997*).

Cutinova Hydra: Cutinova Hydra a new hydro-selective, is highly absorbent, semitransparent, oxygen permeable and flexible. Its use as a modern wound management product is extensive, particularly in moderately exudating wounds. Its properties include selective absorption of wound exudates, maintenance of a moist environment, and protection of wound from trauma.

4. MEDICAL THERAPIES FOR INCREASING VASCULARISATION AND DECREASING OEDEMA

a) Haematorrheological drugs: Which increases the rheological property i.e. fluidity of the blood.

1) Xantinol nicotinate (Nictotinyl xanthinate): It is a compound of xanthine and nicotine acid, both of which are vasodilators. It increases blood flow in many vascular beds and has been promoted for cerebro-vascular disorders and PVDs, but therapeutic benefits are under trial.

Dose : 300-600 mg TDS oral

: 300 mg by i.m or slow i.v. injection.

2) Pentoxiphylline (Oxpentifylline): An analogue of theophylline and a phosphodiesterase inhibitor, it has been shown to increase blood flow in ischemic areas by reducing whole blood viscosity and by improving flexibility of RBCs. The Rheological (dealing with property of flow) action rather than vasodilatation is said to be responsible for improving passage of blood through microcirculation. Thus there are no chances of the steal phenomenon. Oral doses do not affect heart rate, total peripheral resistant and B.P.

Dose : 400mg BD-TDS, 300mg/15ml for slow i.v. injection

b) Clacium Dobesilate:

It decreases platelet aggregation by inhibiting platelet activating factors (PAF) thus prevents the formation of thrombus and the subsequent obstruction

to blood flow. It reduces capillary hyper permeability by increasing the activity of endothelium in nitric oxide synthesis. It decreases erythrocyte aggregation and hyper viscosity of blood by inhibiting lipid per oxidation of erythrocyte membrane. This prevents formation of thrombus.

Dosages : For diabetic microangiopathy 500-1000 mg daily for 4-6 months followed by 500 mg once daily.

ii) Cilostazol

Cilostaxol is a quinoline derivative. Cilostazol and several of its metabolites are cyclic AMP (c-amp) phosphodiesterase III inhibitors (PDE III inhibitors). In Inhibition of phosphodiesterase activity suppresses c-amp degradation with a resultant increase in cAMP in platelets and blood vessels, leading in inhibition of platelet aggregation and vasodilatation, respectively.

Cilostazol reversibly inhibits platelet aggregation induced by a variety of stimuli including thrombin, ADP collagen arachidonic acid epinephrine, and shear stress.

iii) Clopidogrel: It selectively inhibits the binding of the adenosine diphosphate (ADP) to its platelet receptor and the subsequent ADP mediated activation of the glycoprotein GpIIb/IIIa complex, therapy inhibiting platelet aggregation.

iv) Aspirin

It has antiplatelet, analgesic, antipyretic and anti-inflammatory properties. Aspirin inhibits platelet aggregation by irreversible inhibition of platelet cycloxygenase and thus inhibits the generation of thromboxne A_2, a powerful inducer of platelet aggregation and vasoconstriction.

iv] Anti atherogenic drugs:

These drugs include niacin, bile acid-binding resins, HMG-CoA reductase inhibitors (the statins), and clofibrate, gemfibrozil but of clinical significance are the antiplatelet drugs which are manly Aspirin, ticlopidine and clopidogrel.

Ticlopidine: It is a drug which directly interacts with platelet membrane, alters the fibrinogen receptors in such a way that fibrinogen is not able to bind to the activated platelets. As a result platelet aggregation and clot retraction are inhibited.

Dose: 250 mg BD with meals; effect persists several days after discontinuation.

Future trends

The isolation of glycoprotein IIb/IIIa as the fibrinogen receptor on platelet membrane has lead to investigation of a variety of fibrinogen binding antagonists.

Drugs under evaluation are Absiximab, Eptifibatid and Tirofiban.

5. **PATIENT'S EDUCATION [Living with diabetes]**

Today, more than ***(Helfond AE, Matwa P, Chabeli MM, Muller M, European IDDM Policy group)*** 120 millions people world wide are affiliated with diabetes. Millions more display undiagnosed symptoms that lead up to this terrible disease and the incidence of diabetes is on rise. Medical studies show that diabetes has a debilitating effect on the extremities, most often the feet, and of all diabetics admitted to hospitals, over 20% of them because of foot problems. Sadly, many of their problems could have been prevented by proper foot care. The ***American diabetes Association*** reports that 15% of all people with diabetes will eventually develop foot ulcers and that these ulcers frequently become infected and lead to amputation. *50% to 70% of all non traumatic amputations occur in patients with diabetes.* However, professional care, education programs and proper self care can help prevent tragedy and improve the quality of life of people with diabetes.

Families of diabetics

It is very important for the family members of a person with diabetes to understand the basics of diabetics so they can recognize dangers and help

sustain, encourage and remind that persons how to stay healthy.

Self-Care can mean a better life

It is important to remember that aggressive care of the diabetic foot can often offset or alleviate. *Potentially painful and irreversible damages; that will decrease the quality of your life forever.* Common sense, good medical care, and the simple foot care guidelines presented in this booklet can prevent problems that could lead to catastrophic consequences, even amputation.

DAILY CARE

Keep them clean: Wash you feet well with mild soap in medium temperature water every day, Dry carefully, especially between the toes.

Keep them dry: Use a good foot powder sparingly between the toes.

Inspect them daily: Check for blisters, cuts, scratches and discoloration. For dry feet use a very thin coat of lubricating oil cream; *do not put oil or cream between toes.*

Keep them well maintained: Cut toe nails straight across, Consult physician for detailed instruction.

Change socks or stockings every day: Select fitted, seamless socks and do not wear mended socks.

DO NOTs :

Do not smoke

Do not drink alcohol

Do not go barefooted

Do not soak feet

Do not wear sandals with though between toes

Do not sit with legs crossed, or sleep with ankles crosses.

Do not apply heat to feet [*Hot water bottles or heating pads]*

Do not cut corns and calluses. See a foot care specialist

Do not use chemical agents *for removal of corns and calluses*

Do not use corn plasters

Do not walk barefoot on hot surface, like hot sandy beaches or cement around swimming pools.

Self-help, self-care, success

A simple regimen of daily care for a person with diabetes can go a long way toward lessening or eliminating foot problems. The three areas that make up the basic essentials of foot care are:

(1) Washing,

(2) Inspection

(3) Shoe protection.

These three areas come pretty much under your control. So what you do every day is very important to the life and the health success that you can enjoy.

A daily walk, even a short one, helps

Improving the blood supply to the foot can often help to keep it healthy. One way is by walking, which exercises the legs without causing physical stress to the feet. If the weather is bad, walk around the house or go mall walking.

Diabetic foot care for patients:

(Source: Levin, O'Neal, and Bowker, 1993, p.54)

1. Do not smoke
2. Inspect the feet daily for blister, cuts, and abrasions. If patient is unable to reach the plantar surface of the foot, the use of a mirror can aid in observing the plantar surface of the foot. Always check between the toes.
3. Good foot hygiene is a priority. Wash feet daily. Dry the foot carefully, especially between the toes (as maceration can take place).
4. Avoid extremes of temperatures. Test water first with elbows or a thermometer before bathing or soaking feet.

5. If the feet feel cold at night, wear socks. Do not apply hot water bottles, heating pads, or soak the feet in hot water without first testing the temperature. Do not use an electric blanket.

6. Do not walk without footwear

7. Do not walk on hot surfaces such as sandy beaches or on cement around swimming pools.

8. Do not remove or use chemical agents for the removal of corns or calluses. Leave the debridement of corns / calluses to the podiatrists.

9. Do not use adhesive tape on the feet

10. Inspect inside of shoes daily for foreign objects, nail points, torn linings, and rough areas which may causes trauma to feet whilst walking throughout the day.

11. If vision is impaired, ask an assistant (family member, friend) to inspect your feet.

12. For dry feet, use a very thin coat of a lubricating oil or cream. Apply the oil / cream after bathing once the feet are completely dry. Do not apply creams or oils between the toes.

13. Do not wear shoes without hosiery. Hosiery should allow 'breathing' and must not be constrictive in nature.

14. Shoes should be comfortable at the time of purchase. Do not depend on the shoes to stretch out. Shoes should be made out of leather uppers to allow 'breathing'. Purchase shoes from shoe salesman (specialists) who understands diabetic foot problems.

15. Cut nails straight across. Cutting nails down the sides promotes involution of nails which can cause potential infection and complications if the nail penetrates the nail sulcus. If unsure as to how to cut nails, see a podiatrist.

16. Regular visitations of the podiatrist is necessary for routine check ups of neurological and vascular status.

Daily foot inspection is critically important

Remember, if you have insensitive feet, you may be unaware of blisters, sores, cuts and scratches that would be painful to a non-diabetic person. For this reasons, your feet must be inspected daily, including the top and bottom of the foot, the heel and between the toes. Use a hand mirror, or a magnifying glass. Ask a family member to help you if your eyesight is not good or you cannot bend close enough to your feet for careful inspection to them.

6. MODIFIED BOOT THERAPY

After compression and release by a mechanical device including the lower leg and foot called **'BOOT'** has been used for long and has been documented to enhance its circulation and improve healing ***(Neder S, NadashP).***

A number of mechanical deices have been used so far right from use of water to air i.e., pneumatic compression devices for this purpose.

In our system it is very costly to use hydraulic or pneumatic compression devices. We have followed same principal by putting foam around leg and getting the rhythmic regular mild to moderate pressure massage.

Massage being a traditional custom satisfies the psyche and also positively improves the circulation of blood in effected area.

7. MODIFIED SHOE THERAPY

Shoes must always fit comfortably and have adequate width and depth for toes.

Leather shoes easily adapt to the shape of the feet and allow them to "breathe". Athletic shoes and sneakers are usually excellent choices if they are well fitted and provide adequate cushioning.

Footwear can be divided into three broad types:-

(i) Sensible shoes: for patients with minimal sensory loss.

(ii) Readymade stock shoes: for neuroischaemic feet that need protection

along the margins of the foot but that are at greatly deformed and customized.

(iii) Bespoke shoes: containing cradled, cushioned insoles. These are necessary to redistribute the high pressure on the plantar surface of the neuropathic foot.

The diabetic-custom made orthotics will greatly reduce the risk of foot disease. These special orthotics acts as a replacement for the thinning fat pad on the bottom of the feet, thus protecting the skin from excessive bone pressure. This orthotics will also gently support the arch and all the bones and joints of the feet, these are light weight and will fit in all flat shoes these orthotics are the diabetics "ounce of prevention" that may eliminate a "pound of cure" later on.

8. SURGICAL THERAPY TO IMPROVE CIRCULATION.

i) **Sympathectomy**: Sympathectomy is not effective in claudication but occasionally it may relieve ischemic rest pain and ulceration. However it must be recognized that the results of Sympathectomy are very much poorer than those of by-pass surgery or percutaneous transluminal angioplasty. Sympathectomy can only be justified when it is not technically possible to operate or to employ balloon angioplasty.

ii) Surgical by pass grafting: Surgical by pass grafting has evolved as the most widely applicable technique for the treatment of arterial occlusive lesions. It has found broad application in the coronary, abdominal and peripheral vascular bed. In comparison to other techniques such as angioplasty, stenting or endarterectomy, bypass is far less restrictive in terms of the anatomic nature of lesions amenable to treatment.

(a) **Prosthetic grafts**: For bypass of the aortoiliac segment, the favored material is Dacron, for bypass in the femoropopliteal region, if autogenous long saphenous vein (or other veins such as the short saphenous or arm vein) is not

available, PTEE (polytetra fluro ethylene) or glutaraldehyde tanned Dacron-supported human umbilical vein may be employed.

(b)**Venous Grafts:** The most usual and successful conduct is the long saphenous vein used in the institute fashion after disrupting the valves with a valvulotome. However it can be used in reversed fashion. In the lower extremity, long term results with saphenous vein by pass (used in either the in situ or reversed configurations) to below-knee popliteal, tibial, and even pedal arteries have been excellent and serve as the standard of reference for other conducts.

MANAGEMENT OF ACUTE CHARCOT NEUROPATHIC OSTEOARTHROPATHY

Immobilization and reduction of stress are the mainstays of treatment for acute Charcot arthropathy. Many investigators advocate complete nonweightbearing through the use of crutches or other assistive modalities during the initial acute period. While this is an accepted form of treatment, three-point gait may, in fact, increase pressure to the contralateral limb, thereby predisposing it to repetitive stress and ulceration or neuropathic fracture. Following a period of off-loading, a reduction in skin temperature and edema indicates the stage of quiescence at which point the patient progresses into the post-acute phase of treatment. Progression to protected weightbearing is permitted, usually with the aid of some type of assistive device. Through the use of appropriately applied total contact casts or other off-loading modalities (e.g., fixed ankle, walker, bivalved casts, total contact prosthetic walkers, patellar tendon-bearing braces, etc.), most patients may safely ambulate while bony consolidation of fractures progresses. The mean time of rest and immobilization (casting followed by removable cast walker) prior to return to permanent footwear is approximately 4-6 months. There is recent interest in the adjunctive use of bisphosphonate therapy in acute Charcot

arthropathy to help expedite the conversion of the acute process to the quiescent, reparative stage. Similarly, there is interest in managing acute cases with ancillary bone growth stimulation to promote rapid consolidation of fractures. Although promising in theory, neither of these adjunctive treatment to date have been conclusively proven effective through large prospective, randomized clinical trials.

Reconstructive surgery may be considered if a deformity or instability exists that cannot effectively be controlled or accommodated by prescription footwear or bracing. If the arthropathy is identified in its early stages and nonweightbearing is instituted, surgery is usually unnecessary. The consensus of opinion is such that surgery in the acute stage is generally not advisable due to the extreme hyperemia, osteopenia, and edema present. Surgical intervention during the acute phase, however, may be considered in the presence of acute subluxation without osteochondral fragmentation. Refer to the guideline document for further discussion of reconstructive surgery.

- The goal of any surgery undertaken on the Charcot foot is to create a stable, plantigrade foot that may be appropriately accommodated. Surgery is generally undertaken only after radiographic, dermal thermometric and clinical signs of quiescence.
- Following surgery, patients are immobilized until skin temperatures and postoperative edema normalize. As with those treated nonsurgically, following prolonged cast immobilization patients progress to a removable cast walker followed by permanent prescription footwear. Mean times from operation to the wearing of therapeutic shoes have been reported in the range of 27 weeks (7 months). Careful patient selection and management is the rule with these complex diabetic patients since amputation can be an unwanted complication of failed surgical procedures.

AIMS OF STUDY

AIMS OF STUDY

The present study was done in M.L.B. Medical College Jhansi from June 2005 to July 2006 with following aims

A. IN DIABETIC O.P.D.

1. To assess the incidence of foot involvement in diabetic patients
2. Type of predominant presentation:-
   - Diabetic cellulitic foot
   - Peripheral arterial disease
   - Neuropathy
   - Non suppurative phlegmon

B. IN SURGERY WARDS

1. Bacteriology [by culture]
2. Changes in peripheral arteries of limb by peripheral color Doppler study
3. Modes of treatment given

MATERIAL & METHODS

MATERIAL & METHODS

The present study was conducted on patients of diabetes attended diabetic clinic, or admitted in department of surgery in M. L. B. Medical college Jhansi from june 2005 to july 2006 having foot complications.

In diabetic O.P.D. – Study of blood sugar was done

- Categorised to type of foot envolvement depending on predominant envolvement

  -cellulitis

  -neuropathy

  -peripheral vascular disease

  - non specific phlegmon

In surgery indoor wards the detailed history of patients taken with examination and investigations done.

History:A thorough medical and foot history should be obtained from the patient. The following provides guidelines of specific diabetic foot issues that should be addressed:

Global History:

- Diabetes disease duration
- Glycemic management/control
- Cardiovascular, renal, and ophthalmic evaluations
- Other comorbidities
- Social habits - alcohol/tobacco
- Current medications
- Allergies
- Previous hospitalizations/surgeries

Foot-Specific History:

- Daily activity
- Footwear
- Chemical exposures
- Callus formation
- Deformities
- Previous foot surgery
- Neuropathy symptoms
- Ischemic symptoms

Wound/Ulcer History:

- Location
- Duration
- Iniciting event or trauma
- Recurrences
- Infections
- Hospitalizations
- Wound care/off-loading methods
- Patient's compliance/wound response
- Interference with wound care/family or social problems for patient
- Previous foot trauma or surgery
- Edema-unilateral versus bilateral
- Previous or active Charcot joint treatment

A.Physical Examination A general examination of the patient was done including pulse rate, blood pressure, temperature, respiratory rate, pallor, oedema, ecterus, clubbing, cyanosis, hydration status with detailed examination of foot & leg was done.

Recognizing important risk factors and making a logical, treatment-oriented

assessment of the diabetic foot requires a consistent and thorough diagnostic approach using a common language. Without such a method, the practitioner is more likely to overlook vital information and to pay inordinate attention to less critical points in the evaluation. A useful examination will involve identification of key risk factors and assignment into an appropriate foot risk category. Only then can an effective treatment plan be designed and implemented.

B.Clinical Examination

All patients with diabetes presenting to any health care practitioner require a pedal inspection and should receive a thorough foot examination at least once each year. Patients with diabetic foot-related complaints will require detailed evaluations more frequently. The examination should be performed systematically so that important aspects are not overlooked. First, one should grossly evaluate the patient and his or her extremities. Any obvious problem can then receive closer scrutiny with examination. For clarity, the key components of the foot examination are presented below in a bulleted format. Each bulleted item represents an important component of the pedal examination or a significant finding to be noted based on evidence which indicates likely predictors for ulceration. It is assumed that a general medical assessment will be determined including measurements of vital signs.

Vascular Examination

- Palpation of pulses (dorsalis pedis, posterior tibial, popliteal, femoral)
- Venous filling time (normal $\leq$20 seconds)
- Color changes: cyanosis; dependent rubor; erythema
- Presence of edema
- Temperature gradient
- Integumentary changes consistent with ischemia: skin atrophy; nail atrophy; abnormal wrinkling; diminished pedal hair

Neurologic Examination

- Light touch: cotton wool
- Two-point discrimination
- Pain: pinprick
- Temperature perception: hot and cold
- Deep tendon reflexes: ankle, knee
- Clonus testing
- Babinski test
- Rhomberg's test

Musculoskeletal Examination

- Biomechanical abnormalities: orthopedic deformities (hammer toes, bunion(s) or Tailor's bunion(s), flat or high-arched feet, Charcot deformities, iatrogenic deformities (e.g., amputation); limited joint mobility; tendo-Achilles contractures/equinus
- Gait evaluation
- Muscle group strength testing: passive and active, nonweightbearing and weightbearing; foot drop; atrophy - intrinsic muscle atrophy

Dermatologic Examination

- Skin appearance: color, texture, turgor, quality; dry skin
- Calluses: discoloration/subcallus hemorrhage
- Fissures (especially posterior heels)
- Nail appearance: onychomycosis, dystrophic; atrophy, hypertrophy; paronychia
- Presence of hair
- Ulceration, gangrene, infection (Note location, size, depth, infection status, etc.)
- Interdigital lesions
- Tinea pedis

- Markers of diabetes: shin spots - diabetic dermopathy; necrobiosis lipoidica diabeticorum; bullosum diabeticorum; granuloma annulare

Footwear Examination

- Type of shoe
- Fitting of shoe
- Lining wear
- Foreign bodies
- Insoles, orthoses

C.Diagnostic procedures:

Laboratory testing: as indicated:

Hb

TLC, DLC,

Blood urea, creatinine

Random, fasting & post parandial blood sugar,

Urine sugar and ketones

E. C. G.

Pus culture & sensitivity

maging studies:

*x-ray of affected limb

*Vascular procedures (e.g. noninvasive arterial studies color doppler study of peripheral arteries)

Management/Treatment of Diabetic Foot Ulcers

1. Debridement of necrotic tissue (surgical, mechanical autolytic, enzymatic)

2. Pressure reduction (crutches, healing sandal, contact cast, walking brace, foot cast etc.)

3. Wound care (topical saline gauze dressings, antiseptics, special dressings, hyperbaric oxygen therapy (HBO- OXUM), etc.)

4. Management of infection (incision and drainage, empiric and culture directed antibiotics ,soft tissue/ bone/ joint/ resection, amputations

5. Medical management (hyperglycemia, hypertension, nutritional status, renal status)

6. Measures to reduce the risk of ulcer recurrence (regular podiatric care and evaluation; patient preventative education; protective footwear; pressure reduction; surgery to reduce bony prominence/chronic pressure points)

7. Surgical management (curative, ablative, elective)

8. Multidisciplinary consultation and management

Treatment of Diabetic Foot Infections

1. Antibiotic therapy in non-limb threatening infection

    a. Oral agents (amoxicillin/clavulanate, cephalexin, dicloxacillin, clindamycin, levofloxacin)

    b. Parenteral agents (ciprofloxacin, cefotaxime, oxacillin or nafcillin, ampicillin/sulbactam clindamycin)

2. Antibiotic therapy in limb-threatening infection

    ampicillin/sulbactam;

    ticarcillin/clavulanate;

piperacillin/tazobactam;

ceftazidime + clindamycin;

cefotaxime ± clindamycin;

fluoroquinolone + clindamycin;

vancomycin + levofloxacin + metronidazole

3. Antibiotic therapy in life-threatening infection

ampicillin/sulbactam + aztreonam;

piperacillin/tazobactam + vancomycin;

vancomycin + metronidazole + ceftazidime;

imipenem/cilastatin;

fluoroquinolone + vancomycin + metronidazole

Management/Treatment of Charcot Foot

1. Weightbearing restrictions (crutches, wheelchair)
2. Immobilization of foot (splint, cast, removable cast)
3. Special footwear or prostheses (orthopedic or molded foot wear, bracing, insoles)
4. Patient education for prevention of recurrence
5. Surgery

Prevention of Foot Complications

1. Podiatric care
2. Protective shoes
3. Pressure reduction
4. Prophylactic surgery
5. Preventive education

OBSERVATION

OBSERVATIONS

The present study was conducted in M.L.B. Medical College & Hospital, JHANSI (U.P.) to assess the incidence, presentation, macro-vascular involvement, culture study, mode of treatment given & outcome of Diabetic Foot syndrome in Bundelkhand region from June 05 to July 06.

CASE STUDY IN DIABETIC O.P.D.:

As per the hospital records & case study during above mentioned period, total 12901 patients were attended in diabetic O.P.D.

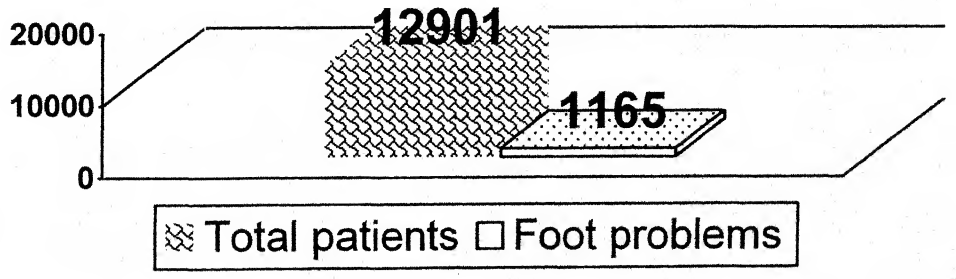
A] Incidence of foot involvement in patients attending diabetic O.P.D.:

Selection criteria: All patients presented in diabetic O.P.D. having any foot complain.

Total patients	Foot problems	percentage
12901	1165	9.03

In our study total 12901 cases observed, in which 1165 cases [9.03%] were having foot problem.

INCIDENCE OF FOOT INVOLVEMENT



B] Type of foot involvement in patients attending diabetic O.P.D.:

Selection Criteria:

1. Cellulitic foot: swollen, hot, tender, infected, with or without gangrene.
2. Neuropathy: H/O tingling, numbness, loss of sensation, decrease power,.

O/E decrease power, decrease nutrition, decrease deep tendon reflexes, loss of touch, pain {pin prick}, and dryness of skin.

3. Peripheral vascular disease: H/O rest pain and/or claudication

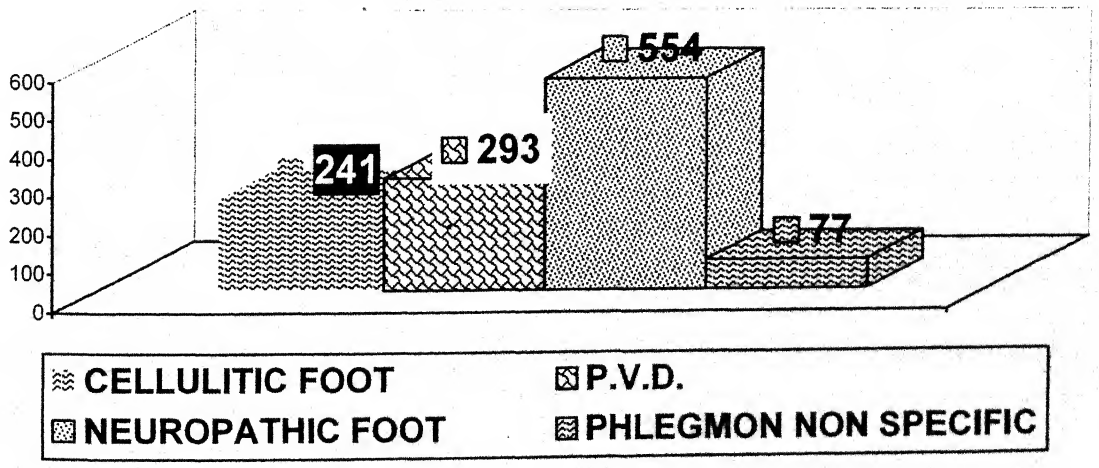
O/E absence of peripheral pulses {dorsalis pedis, post. Tibial, popliteal arteries}, thinning of skin, decrease hairs, loss of subcutaneous fat, brittle nails.

4. Non specific phlegmon: any foot/leg swelling with or with out pain/ tenderness/ redness.

NO.	TYPE OF FOOT INVOLVEMENT	No. of patients	PERCENTAGE OF TOTAL
1.	Cellulitic foot	241	20.69%
2.	Peripheral vascular disease	293	25.15%
3.	Neuropathic foot	554	47.55%
4.	Non specific phlegmon	077	06.06%
5.	Total foot involvement	1165	

In our study we had observed 1165 cases in O.P.D. [DIABETIC], in which most of the complain related to neuropathic changes, the least common type is non specific phlegmon.

PRESENTATION OF DIABETIC FOOT IN O.P.D.



IN SURGERY INDOOR- DIABETIC FOOT PATIENTS' CASE STUDY:

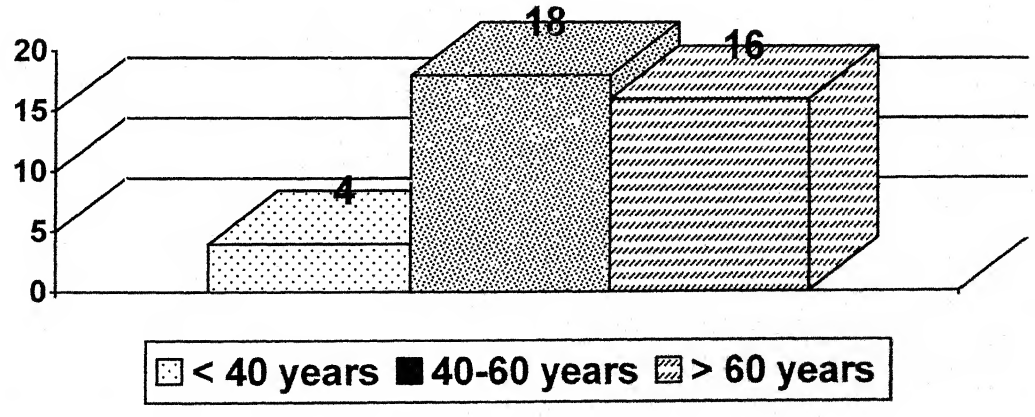
As per records total 38 patients were followed & studied completely during the same period.

1. AGE DISTRIBUTION:

NO.	AGE GROUP	NO. OF PATIENTS	PERCENTAGE%
1.	**≤ 40 years**	**4**	**10.53%**
2.	**40-60 years**	**18**	**47.37%**
3.	**⩾ 60 years**	**16**	**42.10%**
	Total	**38**	

The age distribution of diabetic foot problem in our indoor surgical wards was following:- maximum no. of cases seen in age group of 40-60 years (18 cases)

AGE DISTRIBUTION

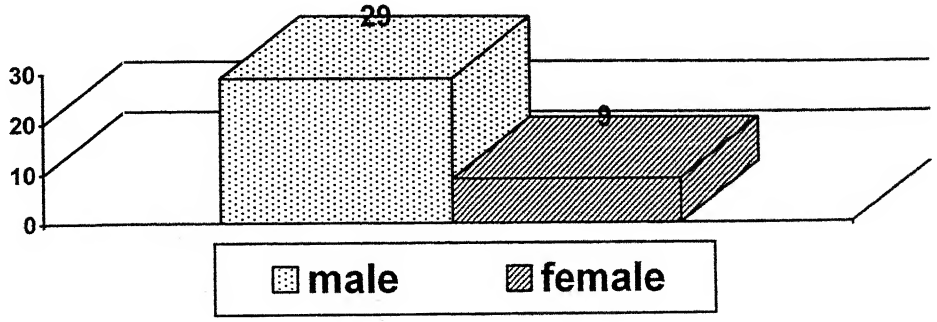


2. SEX DISTRIBUTION:

SEX	NO. OF PATIENTS	PERCENTAGE
MALE	29	76.32%
FEMALE	09	23.68%
TOTAL	38	

As observed in our study 29 cases were male & 09 were female. The male female ratio was 3.22 : 1

SEX DISTRIBUTION

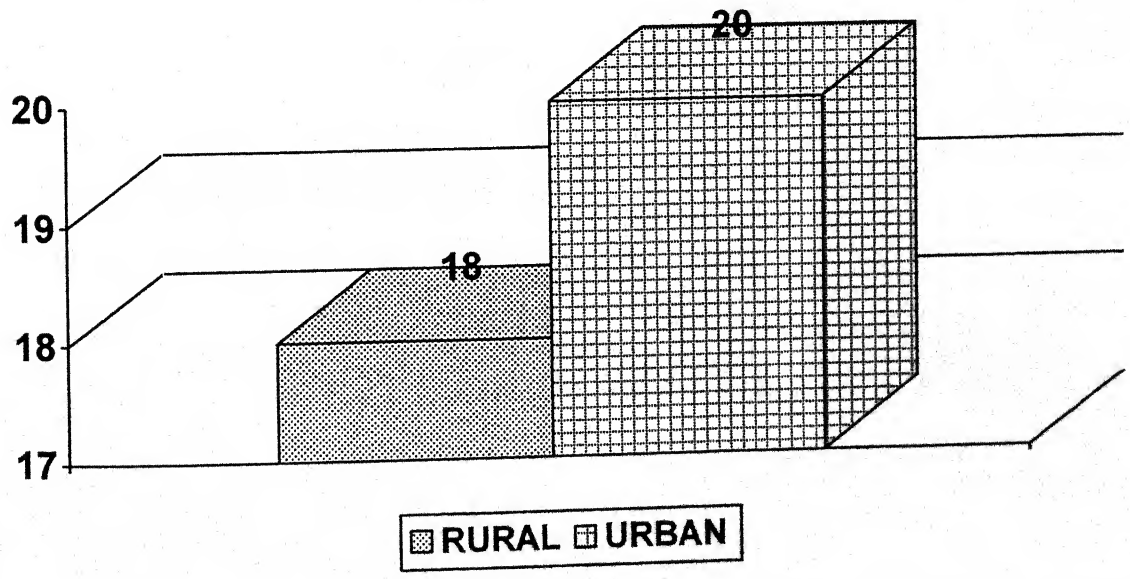


3. RURAL & URBAN DISTRIBUTION:

RESIDENCE	NO. OF PATIENTS	PERCENTAGE%
RURAL	18	47.37%
URBAN	20	52.63%
TOTAL	38	

In this study 18 patients belongs to rural & rest 20 patients were urban. The incidence of diabetes & diabetic foot are much higher in urban population but in our study the difference is not much. It may be because cheaper medical facility in our setup & poverty in the rural area.

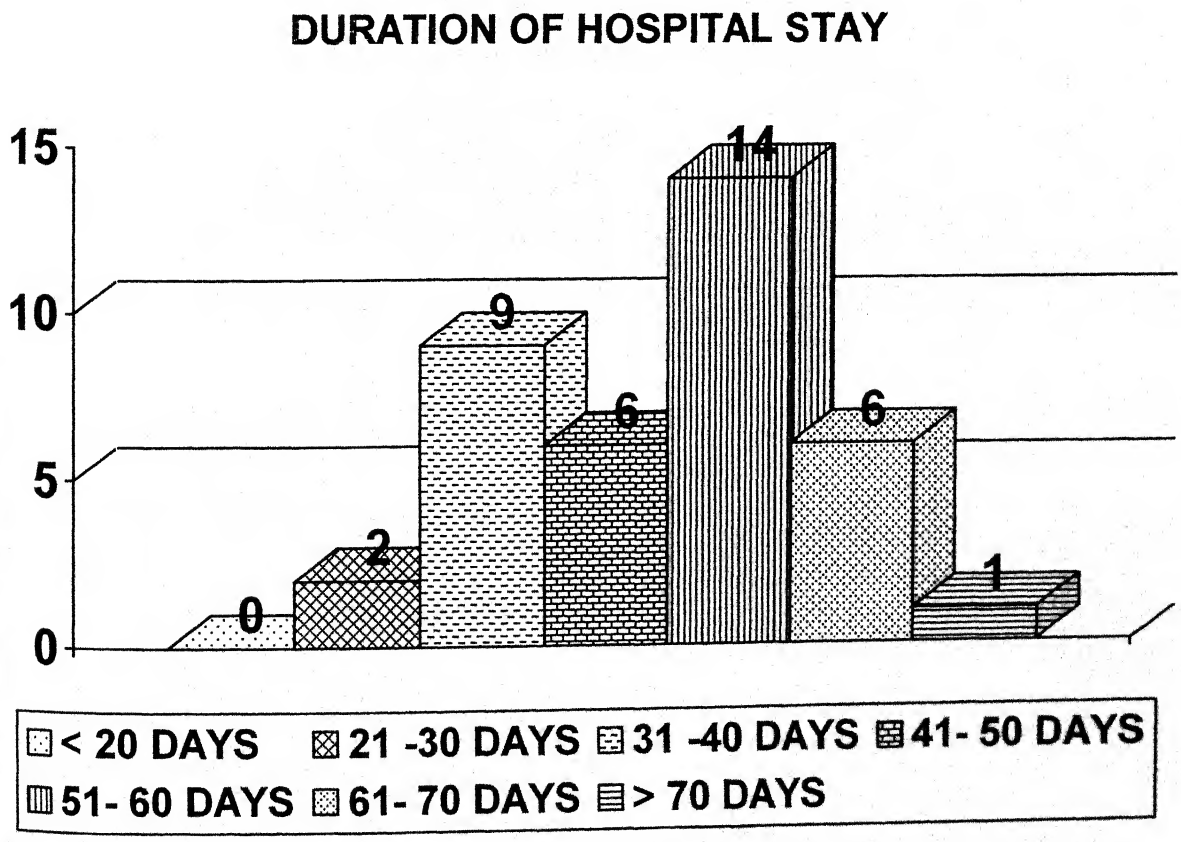
POPULATION DISTRIBUTION



4. DURATION OF STAY IN HOSPITAL:

NO.	DURATION OF STAY	NO. OF PATIENTS	PERCENTAGE%
1.	< 20 DAYS	0	0
2.	21 – 30 DAYS	2	5.26%
3	31 – 40 DAYS	9	23.68%
4.	41 – 50 DAYS	6	15.79%
5.	51 – 60 DAYS	14	36.84%
6.	61 - 70 DAYS	6	15.79%
7.	>70 DAYS	1	2.63%

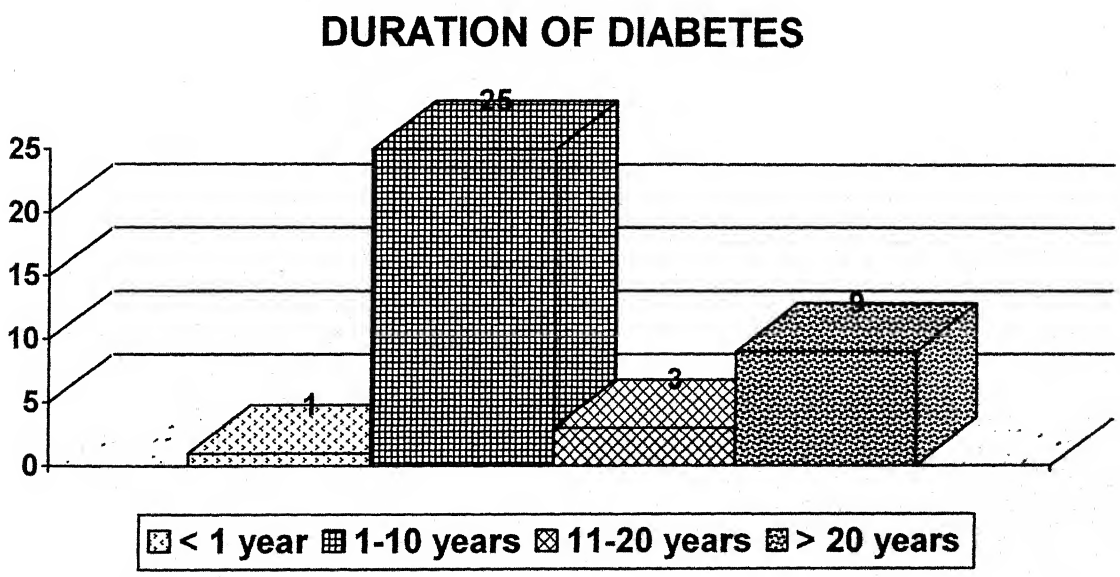
In our study, no one stayed in hospital less than 20 days. The maximum period of stay was more than 70 days [72 days] for only one patient. Most of the patients discharged between 51 – 60 days. The average period of stay is 48.03 days.



5. DURATION OF DIABETES:

No.	Duration of diabetes	No. Of patients	Percentage
1.	< 1 year	1	2.63%
2.	1 – 10 years	25	65.79%
3.	11 – 20 years	3	7.89%
4.	> 20 years	9	23.68%

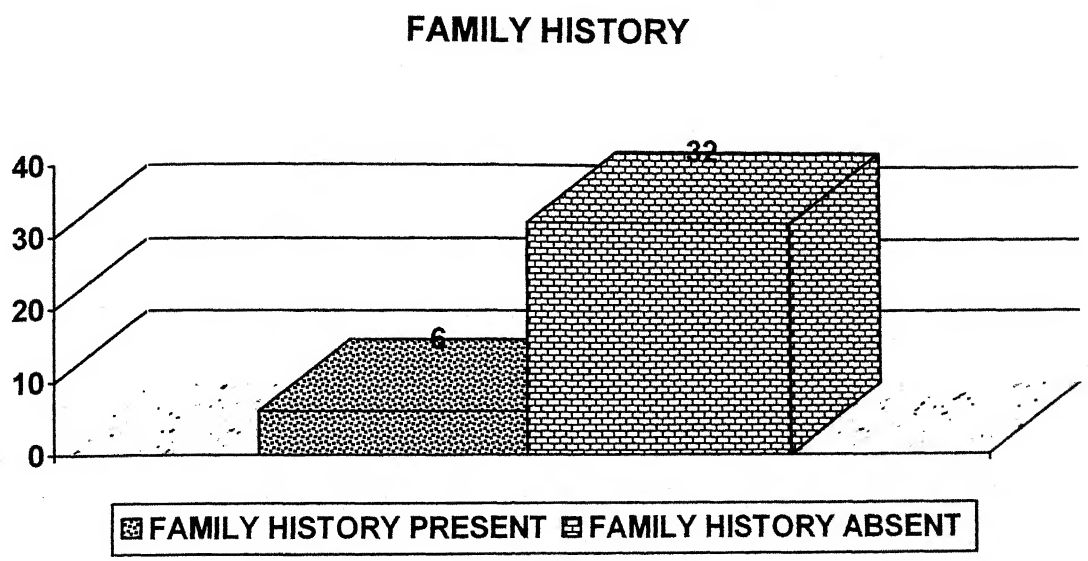
In our study maximum no. of patients with diabetic foot had diabetes 1 – 10 year duration [65.79%], next is > 20 year duration [23.68%].



6. FAMILY HISTORY:

No.	Patients	No. of patients	Percentage
1.	Family history present	6	15.79%
2.	Family history absent	32	84.21%

In our study majority of cases were not having positive family history (84.21%)



7. DOMINANT PRESENTATION OF DIABETIC FOOT in SURGERY WARDS:

Selection Criteria:

1. Cellulitic foot: swollen, hot, tender, infected, with or without gangrene.

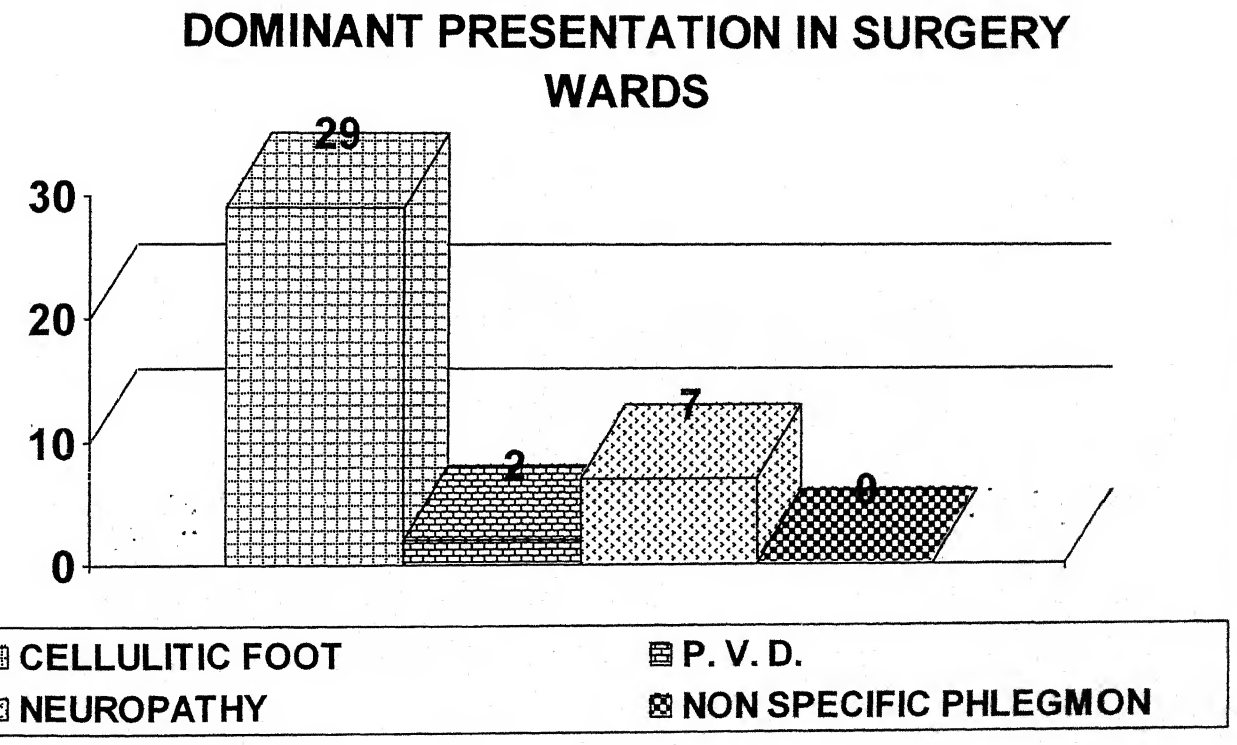
2. Neuropathy: H/O tingling, numbness, loss of sensation, decrease power,
   O/E decrease power, decrease nutrition, decreases deep tendon reflexes, loss of touch, pain {pin prick}, temperature {hot/cold} and dryness of skin.

3. Peripheral vascular disease: H/O rest pain and/or claudication
   O/E absence of peripheral pulses {dorsalis pedis, post. tibial, popliteal arteries}; thinning of skin, decrease hairs, loss of subcutaneous fat, brittle nails.

4. Non specific phlegmon: any foot/leg swelling with or with out pain/ tenderness/ redness.

NO.	DOMINANT PRESENTATION	NO. OF PATIENTS	PERCENTAGE
1.	Diabetic cellulitic foot(septic foot)	29	76.31%
2.	Peripheral vascular disease	2	5.26%
3.	Neuropathy	7	18.42%
4.	Non suppurative phlegmon	0	0%

In this study the most common predominant presentation is diabetic cellulitic foot, for which patient admitted in our wards.

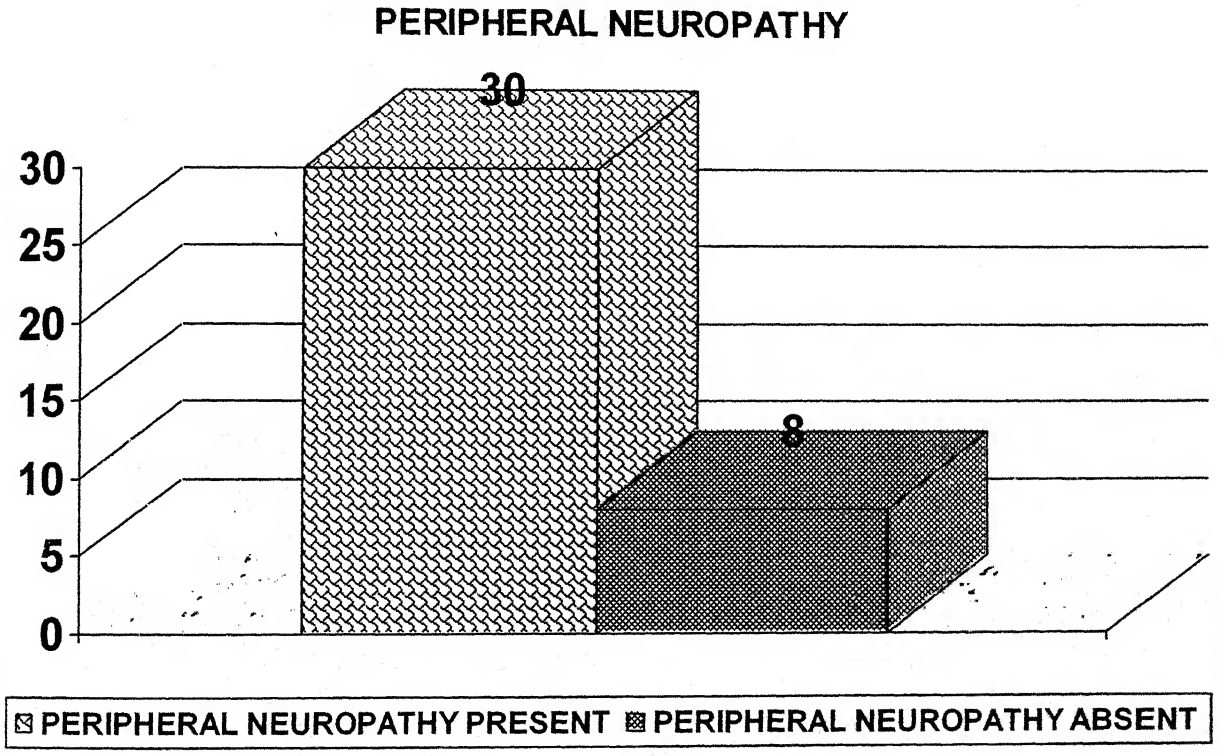


8. PERIPHERAL NEUROPATHY IN INDOOR DIABETIC FOOT PATIENTS:

Selection criteria: H/O tingling, numbness, loss of sensation, decrease power, O/E decrease power, decrease nutrition {bulk of muscles}, decrease deep tendon reflexes, loss of touch, pain {pin prick}, temperature {hot/cold}, two point sensations (5 mm. apart at least) & joint position sensations and dryness of skin.

NO.	CASE	NO. OF PATIENTS	PERCENTAGE
1.	Peripheral neuropathy present	30	78.94%
2.	Peripheral neuropathy absent	8	21.06%

In this study most of the indoor diabetic foot patients were suffering from neuropathy (78.94%).



9. PERIPHERAL VASCULAR DISEASE IN DIABETIC FOOT PATIENTS (INDOOR):

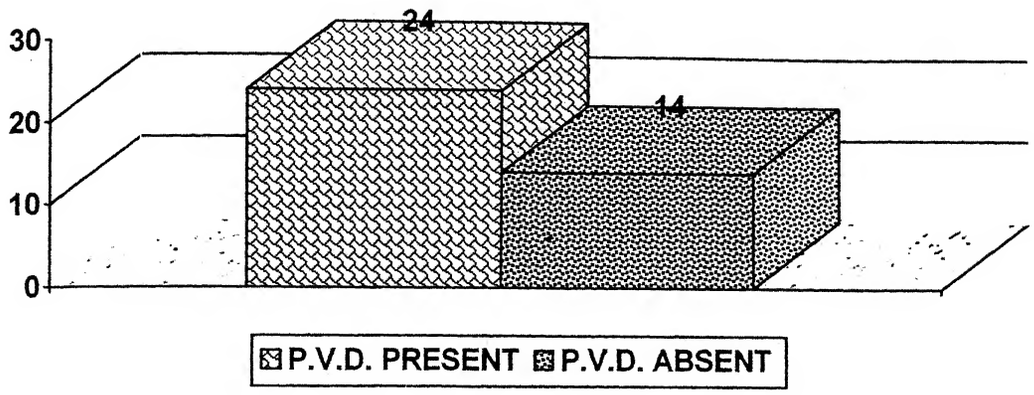
Selection criteria: H/O rest pain and/or claudication

O/E absence of peripheral pulses {dorsalis pedis, post. tibial, popliteal arteries}, thinning of skin, decrease hairs, loss of subcutaneous fat, brittle nails.

NO.	CASE	NO. OF PATIENTS	PERCENTAGE
1.	P.V.D. PRESENT	24	63.16%
2.	P.V.D. ABSENT	14	36.84%

In this study 63.16% patients who were admitted in our institution for diabetic foot problem, had peripheral vascular insufficiency.

PERIPHERAL VASCULAR DISEASE

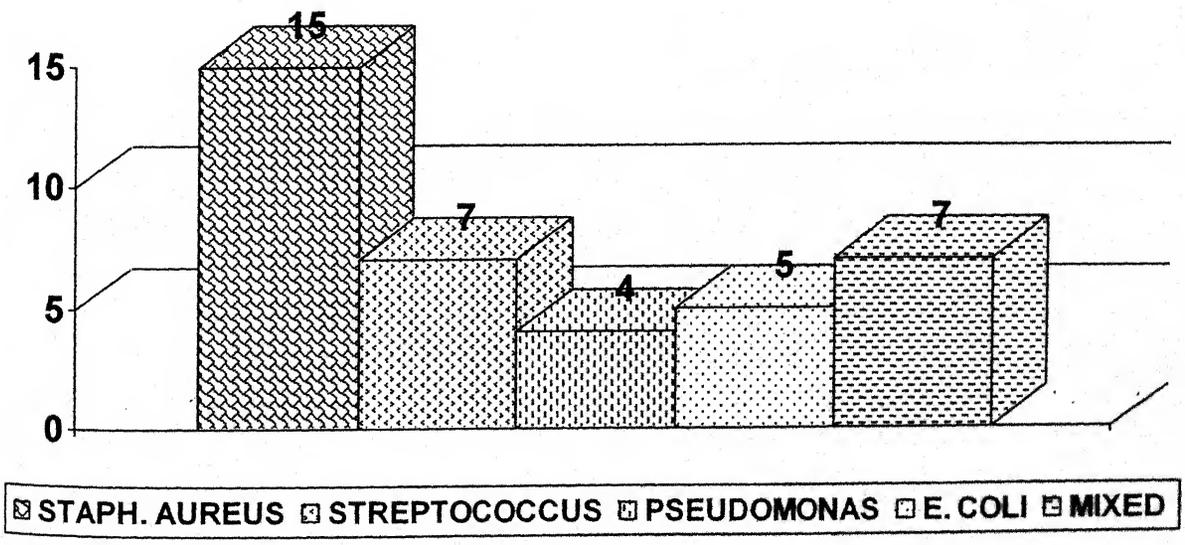


10. BACTERIOLOGIC STUDY:

No.	Bacteria	No. of patients	Percentage
1.	Staph. aureus	15	39.47%
2.	Streptococcus pyogens	7	18.42%
3.	Pseudomonas	4	10.53%
4.	E. coli	5	13.16%
5.	Mixed	7	18.42%

In this study Staph. aureus was most common organism[39.47%], next was streptococcus[18.42%] & mixed infection { mainly with E. coli, Klebsiella & streptococcus}.

ORGANISM



11. SYSTEMIC COMPLICATIONS:

Selection criteria:

a. Cardiopathy: coronary heart disease, E.C.G. changes showing cardiomegaly, C.H.F. & conduction abnormalities.

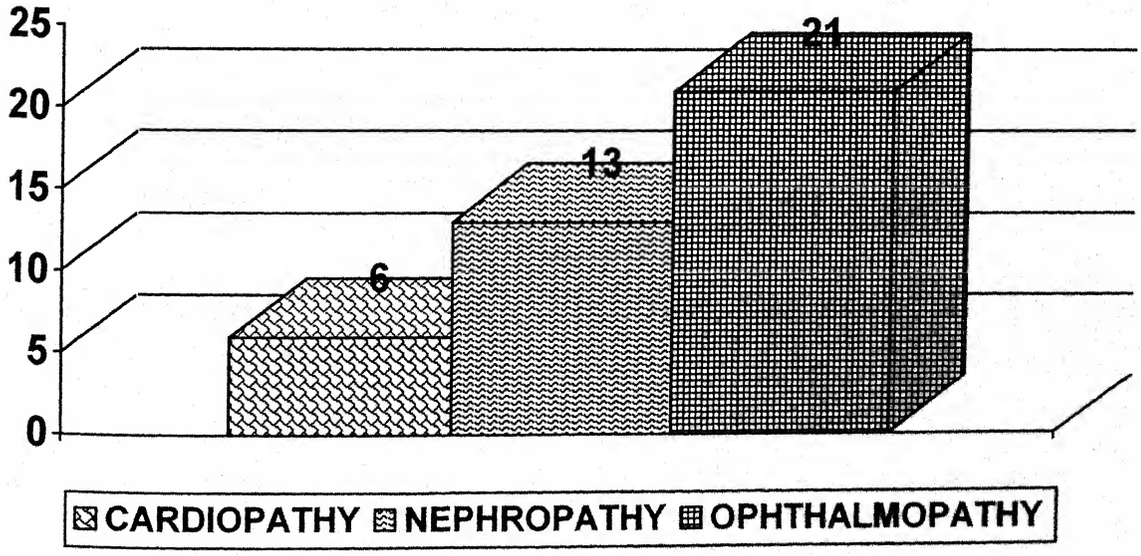
b. Nephropathy: renal enlargement, microalbuminuria, increase serum creatinine level, increase blood urea level.

c. Ophthalmopathy: microanurysm, hard & soft exudates, neoangiogenesis, diffuse small hemorrhage around macula. Secondary glaucoma, hard exudates around macula.

No.	Complications	No. of patients	Percentage
1.	Cardiopathy	6	15.79%
2.	Nephropathy	13	34.21%
3.	Ophthalmopathy	21	55.26%

In our study of cases Ophthalmopathy was most common {55.26%}, next was nephropathy {34.21%}.

SYSTEMIC COMPLICATIONS



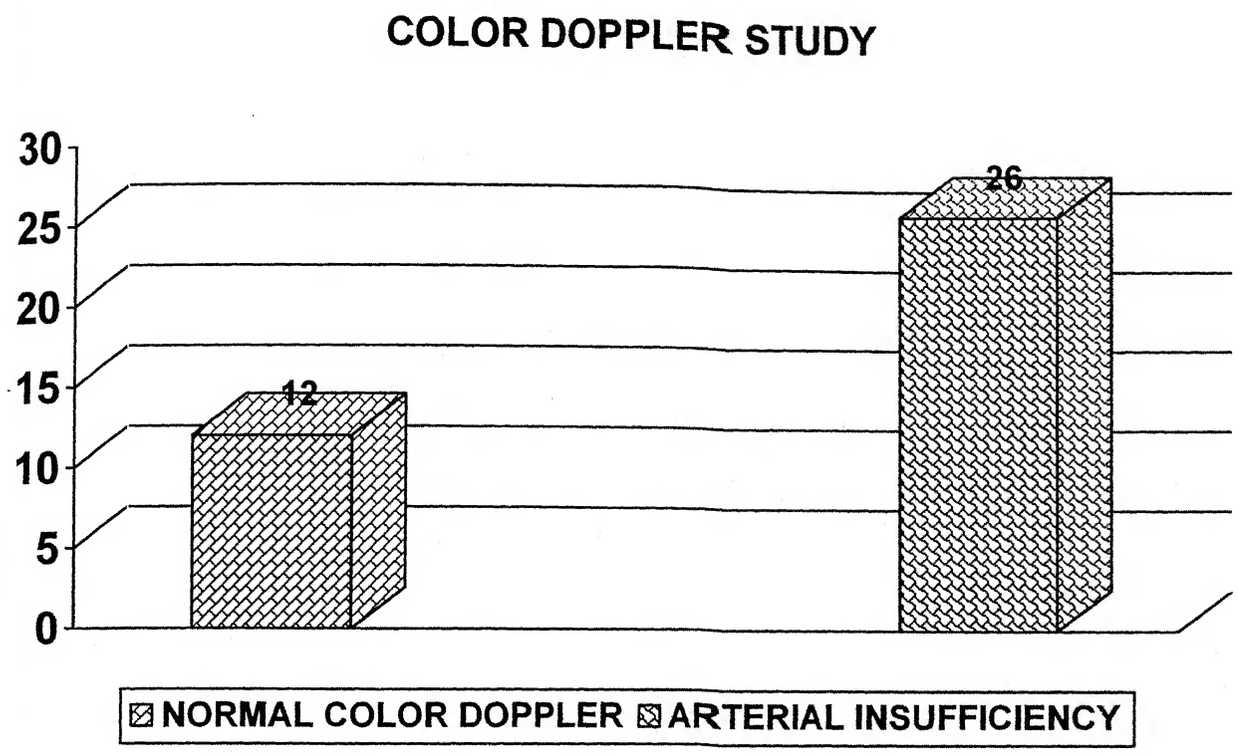
12. COLOR DOPPLER STUDY FOR PERIPHERAL ARTERIES IN LOWER LIMB:

Selection criteria: *{Elsevier-Mosby vol.1- DIAGNOSTIC ULTRASOUND Carol M. Rumack, Stephanine R. Wilson. 3ed ed. Pg. 1003 }*

1. Increase peak systolic velocity > 200 cm./sec.{stenosis at least 50% diameter}.
2. Increase peak systolic velocity ratio 2 or more{ stenosis 50% or more}.
3. Decrease peak systolic velocity with biphasic (high resistance) or monophasic (high resistance).
4. Absent flow signals (false positive for occlusion/false negative for stenosis).

No.	Color Doppler study	No of patients	percentage
1.	Normal color Doppler study	12	31.58%
2.	Color Doppler suggestive of arterial insufficiency	26	66.42%

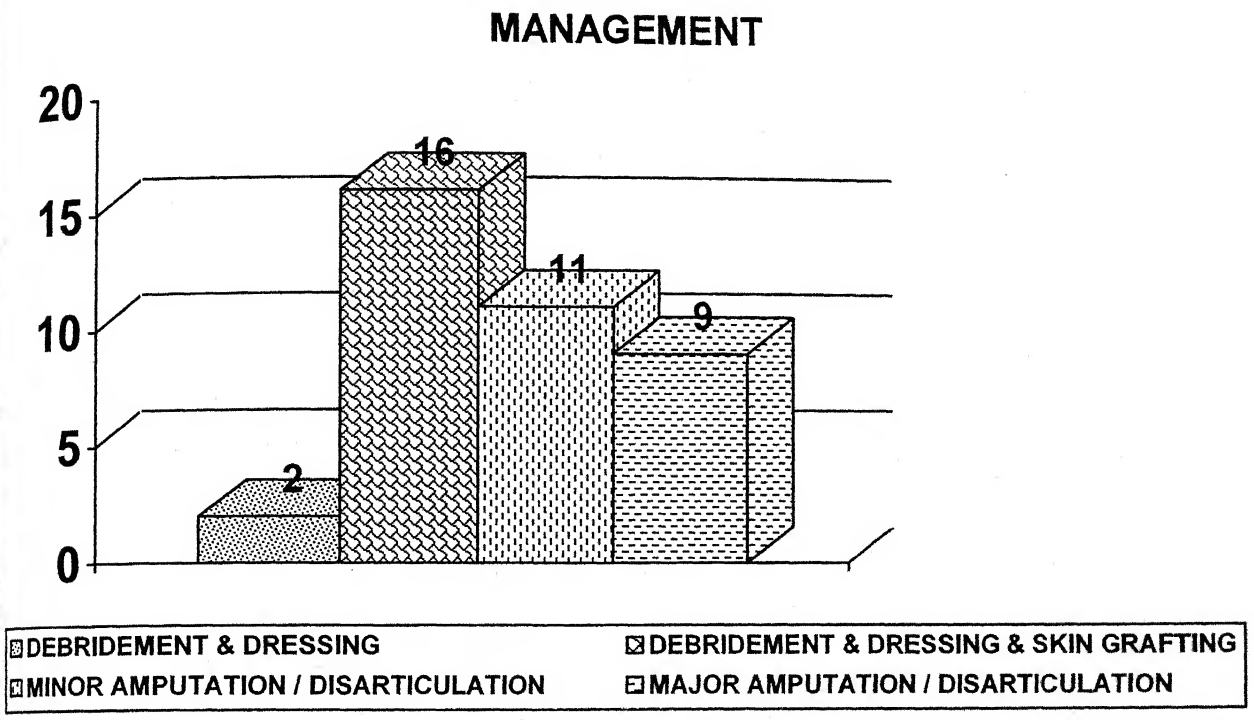
In our study, color Doppler imaging for peripheral vessels detects vascular insufficiency in 66.42% cases of diabetic foot, Rest of the 31.58% cases was normal on color Doppler study.



13. MANAGEMENT OF CASES {SURGICAL}:

No.	Management given	No. of patients	percentage
1.	Debridement (s) & dressing alone	2	5.26%
2.	Debridement (s), dressing & skin grafting	16	42.11%
3.	Debridement & Minor amputations/ disarticulations	11	28.95%
4.	Debridement & Major amputations/ disarticulations	9	23.68%

All the patient had requires surgical treatment. The commonest mode of treatment given was debridement & skin grafting {42.11 %} followed by minor amputation / disarticulation{ of toes & metatarsals}{28.95%}



14. **MORTALITY:** In our study no mortality was noted from June 05 to July 06.

DISCUSSION

DISCUSSION

In this study,12,901 diabetic patients were attended in diabetic O.P.D. department of medicine & 38 patients with diabetic foot admitted in surgery department of M.L.B. medical College, Jhansi, from June 05 to July 06, were studied under the following heads :-

A] O.P.D. PATIENTS

1} INCIDENCE OF FOOT PROBLEMS:

- In a study at ***S.C.B. Medical College, Cuttack*** the incidence of diabetic foot was 10%.
- ***Scott D. Ramsay et.al.*** in their study showed that among 8,905 patients identified with diabetes, 514 developed a foot ulcer over 3 years of observations (i.e. incidence was 5.8%). The incidence of ulcers in this cohort of patient with diabetes was nearly 2% per year.
- ***Branko Novak ,Zeljko Metelko, Nikica Car*** in their study reported about 15% of diabetes patient affected by foot problems.
- In another study, held in ***Deptt. Of medicine, Manchester Royal infirmary, UK,*** the cumulative life time incidence of foot ulcerations in diabetic patients was a high as 15%.
- The prevalence of diabetic foot has been estimated to be 3-8%.***{Ebskov B., Ebskov L.; Diabetologica;vol.39;number 12/nov.1996;pg.1607-10}.***
- In another study held in ***Deptt. Of Medicine, College of Health Sciences University of Nairobi, Kenya (2003 Jan)***, the incidence of foot ulcers in diabetic patients was 4.6%.
- The incidence of diabetic foot in patients of Diabetes mellitus in our study is 9.03% (i.e. 1165 out of 12901). This incidence is comparable to Indian study at Cuttack, but show higher incidence than other international studies. The higher

incidence of Diabetic foot in Diabetic patients in Bundelkhand region is due to poor foot care, illiteracy and inadequate medical facilities in this socio-economically backward region.

2} DOMINANT PRESENTATION:

Dominant Presentation of Diabetic foot:-

- In a study at ***Cuttack hospital,*** Dominant presentations of Diabetic foot were as neuropathy (50%). Septic diabetic foot (16%) and PVD (26.6%).
- In their study, ***Pecoraro et. al. (1991)*** showed that PVD was associated with 62% of non-healing foot ulcers in diabetes as seen in the western population and presentation as septic diabetic foot in 26%.
- It has reported that 60% of patients with diabetes have some form of neuropathy ***{www.niddk.nih.gov}.***
- ***Branko Novak ,Zeljko Metelko, Nikica Car*** reported peripheral neuropathy in 80% cases.
- A population based study in North of England showed that 42% of Type II Diabetes had clinical evidence of neuropathy another study at ***Flinders University Northern territory Clinical School and Royal Darwin Hospital, Darwin, Australia*** showed that peripheral neuropathy was dominant presentation of diabetic foot in about 63% cases.
- ***S.C.N.A. Vol.78; June 1998; pg-393*** reported virtually every diabetic with diabetes for more than 10-15 years has some evidence of neuropathy.
- In our study, dominant presentation in O.P.D. {diabetic} was peripheral neuropathy (47.55%), where as peripheral vascular disease was observed in 25.15% cases. Still sepsis, in general, is a common problem {20.69%} in rural areas; and Diabetics are the more susceptible to septic complications in general and in foot especially.

B] SURGERY INDOOR PATIENTS

1} AGE DISTRIBUTION:

- In a study at ***Medical College, Cuttack,*** maximum number of cases was seen in 6th decade.
- In another study held ***at Diabetic research centre, Royapuram, Chennai, India (2000 Mar.)*** age at presentation was 44-64 years.
- ***Pecararo RE et al,*** in their study showed that among hospital discharges for foot ulcers during 1983-90 the highest percentage was in persons aged 45 to 64 years.
- According to another study, ***Qari FA, Akbar D King Abdulaziz University Hospital, Saudi Arabia,*** Majority of patients with diabetic foot were male above 50 years of age group.
- The average age of presentation were 71 in Germany, 56 in India & 51 in Tanzania ***{Morbach S, Lutale JK, Vishvanath V et al. Diabet. Med. 2004 jan;21(1):91-5}.***
- In our study also the age of presentation of Diabetic foot was 40-60 years{47.37%}, & >60years {42.10%}. Thus in most of the studies, the average of presentation of diabetic foot were 40-60 year.

2} SEX DISTRIBUTION:-

- In a study at ***medical college Cuttack*** the male and female ratio was 9:1 (i.e. 90% were male).
- According to another study held at ***Diabetic research centre Royapuram, Chennai India (2000 Mar.)*** male to female ratio was 1.54:1 (i.e. incidence was higher is males).
- In a retrospective study conducted at ***Carilion Raaroka Community Hospital in Virgina USA by Stepha L. et al.*** it was shown that 53% male & 47% females had diabetic foot ulcer i.e. incidence was significantly higher in males.

- In our study, percentage of male & females with diabetic foot are 76.32% and 23.68% respectively. Thus in most of the studies, the incidence of diabetic foot is higher is males, may be due to comparatively more outdoor activity in males.

3. RURAL AND URBAN DISTRIBUTION:

- In our study, 47.37% patients belonged to rural area; & a 52.64% patient from urban area. This incidence in rural & urban area was almost equal in this study.

4. DURATION OF DIABETES AT PRESENTATION OF FOOT COMPLICATION:-

- In a study average diabetes duration until the onset of the initial foot lesion was 14 years in Germany,12 years in India & only 5 years in Tanzania*{Morbach S, Lutale JK, Vishvanath V et al. Diabet. Med. 2004 jan;21(1):91-5}*.
- In a study at ***medical college, Cuttack*** the incidence of foot complications in Diabetic patients was 50% within 5 year duration.
- According to another study held at ***Diabetic foot clinics, Deptt. of internal medicine, Marienkrankenhaus, Soest, Germany*** average diabetes duration until the onset of initial foot lesion was 14 years in Germany, 12 years in India & only 5 years in Tanzania.
- In our study, 65.79% patients (i.e. 25 out of 38) had diabetes of less than 10 years.

5. DOMINANT PRESENTATION OF DIABETIC FOOT IN SURGERY WARDS:-

- In a study at ***Cuttack hospital,*** Dominant presentation of Diabetic foot was as neuropathy (50%). Septic diabetic foot (16%) and PVD (26.6%).
- In their study, ***Pecoraro et. al (1991)*** showed that PVD was associated with

62% of non-healing foot ulcers in diabetes as seen in the western population and presentation as septic diabetic foot in 26%.

- ***Oyibo S.O.;Jude E.B.;Tarawneh I. et al.Diabetic Medicine; vol.18; pg.133 / feb2001*** reported majority (67%) diabetic foot ulcers are neuropathic.
- Peripheral vascular disease was 48% in Germany, 12% in Tanzania & 13% in India***{Morbach S, Lutale JK, Vishvanath V et al. Diabet. Med. 2004 jan;21(1):91-5}***.
- A population based ***study in North of England*** showed that 42% of Type II Diabetes had clinical evidence of neuropathy another study at ***Flinders University Northern territory Clinical School and Royal Darwin Hospital, Darwin, Australia*** showed that peripheral neuropathy was dominant presentation of diabetic foot in about 63% cases.
- In our study, dominant presentation of diabetic foot was septic foot (76.31%) where as peripheral neuropathy was observed in 18.42% cases & peripheral vascular disease in 5.26% cases. But cases of diabetic septic foot having associated problem of peripheral neuropathy in 78.94% cases & peripheral vascular disease in 63.16% cases. Still sepsis, in general, is a common presenting problem in this area; and Diabetics are all the more susceptible to septic complications in general and in foot especially. The involvement of foot as compared to any other part of body is higher in world at large. In our rural dominant population, the foot involvement is still higher because people walk bare footed and is prone to various kinds of trauma everyday. Over & above, there is lack of foot care, illiteracy and inadequate medical facilities in this group of people in Bundelkhand region.

6. SYSTEMIC COMPLICATIONS:-

- In western countries diabetes is most common cause of blindness in people

aged 20-60 years. Approximate 80-90% of patients shows some degree of retinopathy 20 years of diagnosis; 10-20% of patients with type 2 diabetes, however, may have retinopathy at the time of diagnosis*{fast facts-diabetes mellitus-oxford-Campbell I.W., Lebovitz Harold, 2000}*.

- Diabetic nephropathy develops in 6-27% of type 1 diabetic patients & 10-33% of type 2 diabetes*{fast facts-diabetes mellitus-oxford-Campbell I.W., Lebovitz Harold, 2000}*.
- In our study Cardiopathy present in 15.79%, nephropathy in 34.21% & Ophthalmopathy in 55.26% cases. In this study most of the cases associated with Ophthalmopathy.

7. BACTERIOLOGY:-

- In a study at ***Cuttack hospital,*** dominant infective organisms were Staph. aureus (35.7%) Proteus (25%) and pseudomonas (14.3%) in Septic diabetic foot.
- In a study at ***Diabetic Research Centre Royapuram, Chennai, India,*** aerobic pathogens (66.8%) were the most common organisms isolated from infected diabetic foot.
- ***Qari FA, Akbar D, in their study at King Abdulaziz university hospital, Jeddah. Saudi Arabia,*** found that Proteus and Pseudomonas were the most common organisms isolated from infected diabetic foot.
- ***Rooh-Ul-Muqin, Ahmed M, Griffins in their study at Khyber Teaching Hospital, Peshwar, Pakistan*** observed that Staphylococcus was the commonest organism isolated from cultures obtained from infected diabetic foot.
- ***Zafar, A. Ayub Medical College, Abbottabad,*** observed that Staphylococcus aureus (54%) was the most common organism isolated from cultures obtained from infected diabetic foot.
- In our study, predominant infecting organism was Staph. aureus (39.47% cases) followed by mixed infection (18.42%).

8. COLOR DOPPLER STUDY FOR PERIPHERAL VASCULAR INVOLVEMENT:

- In a study by Debkaran Bhavesh, Minhas SS, Bharadwaj Rajeev in Indira Gandhi Medical College, Shimla, they reported 76% patients were found to have involvement of peripheral vessels.
- In our study all the 38 patients were examined for vascular involvement by color Doppler ultrasonography. The 66.42% patients {26 out of 38} show vascular involvement.

9. MODE OF TREATMENT GIVEN:

A) DRESSING, DEBRIDEMENT & SKIN GRAFTING:

- ***Qari FA, Akbar D at King Abdulaziz University, Hospital, Jeddah,Saudi Arabia*** showed that 65% of patients with diabetic foot lesions need dressing & debridement.
- ***Caravauai C. D.E. Gislio R. et. al. Oct. 2003***. They achieved a complete ulcer healing in 65.3% cases in treated group and 49.6% in control group after weekly assessment, aggressive debridement, and adequate pressure release.
- In our study, all patients with diabetic septic foot were treated by incision & drainage and aggressive debridement either in single setting or in multiple settings.

B) AMPUTATIONS:

- ***Scott D. Ramsay et. al.*** in his study reported a lower extremity amputation rate of 11.2% in patients with diabetic foot lesions.
- ***Moulik PK et. al.*** University Hospital, Aintree, Liverpool, U.K., reported five year amputation rate for ischemic (29%), neuroischaemic (25%) and neuropathic (11%) ulcers.
- Approximately 40-60% of all amputation of lower extremity are performed in

patients with diabetics.*{Ebskov B., Ebskov L.; Diabetologica;vol.39;number 12/nov.1996;pg.1607-10}.*

- ***Qari FA et. al.*** in his study reported a major amputation rate of 23.5% in patients with diabetic foot lesions.
- **DEPT. of surgery, Derer's University Hospital, Brastislava, Slovakia.** Reported 50% patients of diabetes need amputations due to complications mainly gangrene.
- ***O'Rourkee I, Heard S, et. al.*** in his study found that 37% and 23% of patients with diabetic foot lesions required minor & major amputations respectively.
- ***Vega Daniel, West Kristine, Tellado Jose M.*** reported 30-50% of diabetes with critical limb ischemia requires amputation.
- In our study the amputation rate is significantly higher due to high rate of infection in Indian set up. Amputations were necessary, beneficial, cost effective & life saving in cases of extensive gangrene in diabetic foot. In our study, amputation was done in 52.63% cases {in form of minor (28.95%) or major (23.68%) amputations}.

10. DURATION OF HOSPITAL STAY:

- In our study the maximum number of patients stayed in hospital for 51-60 days {36.84%} followed by 31-40 days {23.68%}.the average stay is of 49.71 days.

11. MORTALITY:-

- ***Moulik P K et al,*** in his study reported five year mortality rate of 45% for Neuropathic, 18% for Neuroischemic and 55% for ischemic ulcers.
- ***Wang et. al.*** in his study concluded that considerable international differences were found not only in mortality rate following amputations but they also very according to the type of diabetes. In his opinion, IDDM have a high mortality rate.
- In a study {by ***Oyibo S.O.;Jude E.B.;Tarawneh I. et al.Diabetic Medicine; vol.18;***

pg.133/feb2001}4% patients died.

- In our study, mortality rate was NIL. Mortality rate in patients with diabetic foot lesions have reduced significantly due to improved medical care and antibiotics; but in our study it was nil because follow-up of patients was only for one year & poor patient compliance for follow-up.

CONCLUSION

COCLUSION

These conclusions are based on 12,901 patients who had attended diabetic O.P.D. & 38 patients who had been admitted in surgical wards, from June 2005 to July 2006 in M.L.B. Medical College, Jhansi.

A. DIABETIC O.P.D. CONCLUSION:

1. In present study, the incidence of foot problem in patients' who had attended diabetic O.P.D. was 9.03%.
2. The dominant presentation in O.P.D. patients was peripheral neuropathy {47.55%}, peripheral vascular disease {25.15%} followed by cellulites {septic} foot {20.69%}.

B. SURGERY WARDS CONCLUSION:

1. The maximum number of patients with foot problems was observed in age group of 40-60 years {47.37%} followed by > 60 years group {42.10%}.
2. The male: female ratio was 3.22: 1.
3. The average duration of stay in hospital was 49.71 days. The most of the patients stayed in hospital for 51-60 days {36.84%} followed by 31-40 days {23.68%}.
4. The population distribution is almost equal. The patients presented in our hospital 47.27% belongs to rural & 53.63% belongs to urban area.
5. Majority of cases had diabetes duration history of 1-10 years {65.79%}.
6. The dominant presentation in surgery wards was cellulites {76.31%} followed by peripheral neuropathy {18.52%}.
7. Family history was positive for diabetes in admitted patients only in 15.79% cases.
8. The bacteriological study showed Staph. Aureus was most common infective organism in presented cases {39.47%} second one was

streptococcus pyogens {18.42%} & mixed infection {18.42%}.

9. Systemic complication which most commonly associated was Ophthalmopathy {55.26%}, next was nephropathy {34.21%}.
10. Color Doppler study showed 66.42% cases had some form of vascular insufficiency.
11. Most of the cases responded to dressing, debridement & skin grafting {42.11%}. Amputation/ disarticulation of one or more toes or metatarsals required in 28.45% cases. Major amputations/disarticulations [of ankle, above ankle, below knee, knee, and thigh] required in 23.68% cases.
12. In our study period no mortality was recorded.

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